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EDUCATIONAL NUMBER

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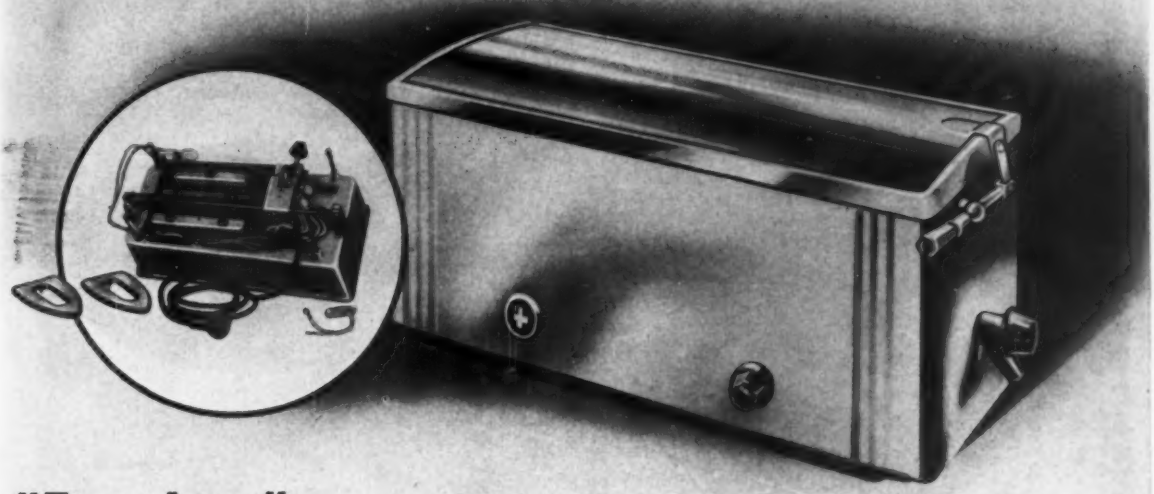

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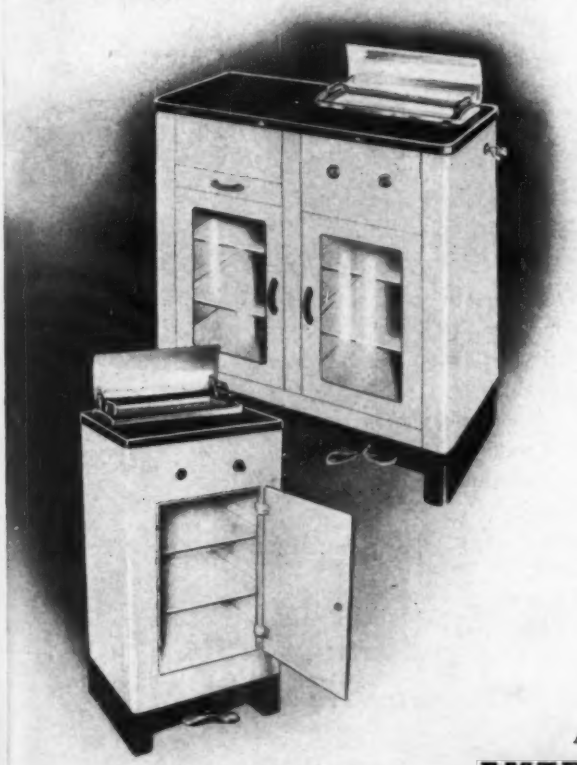

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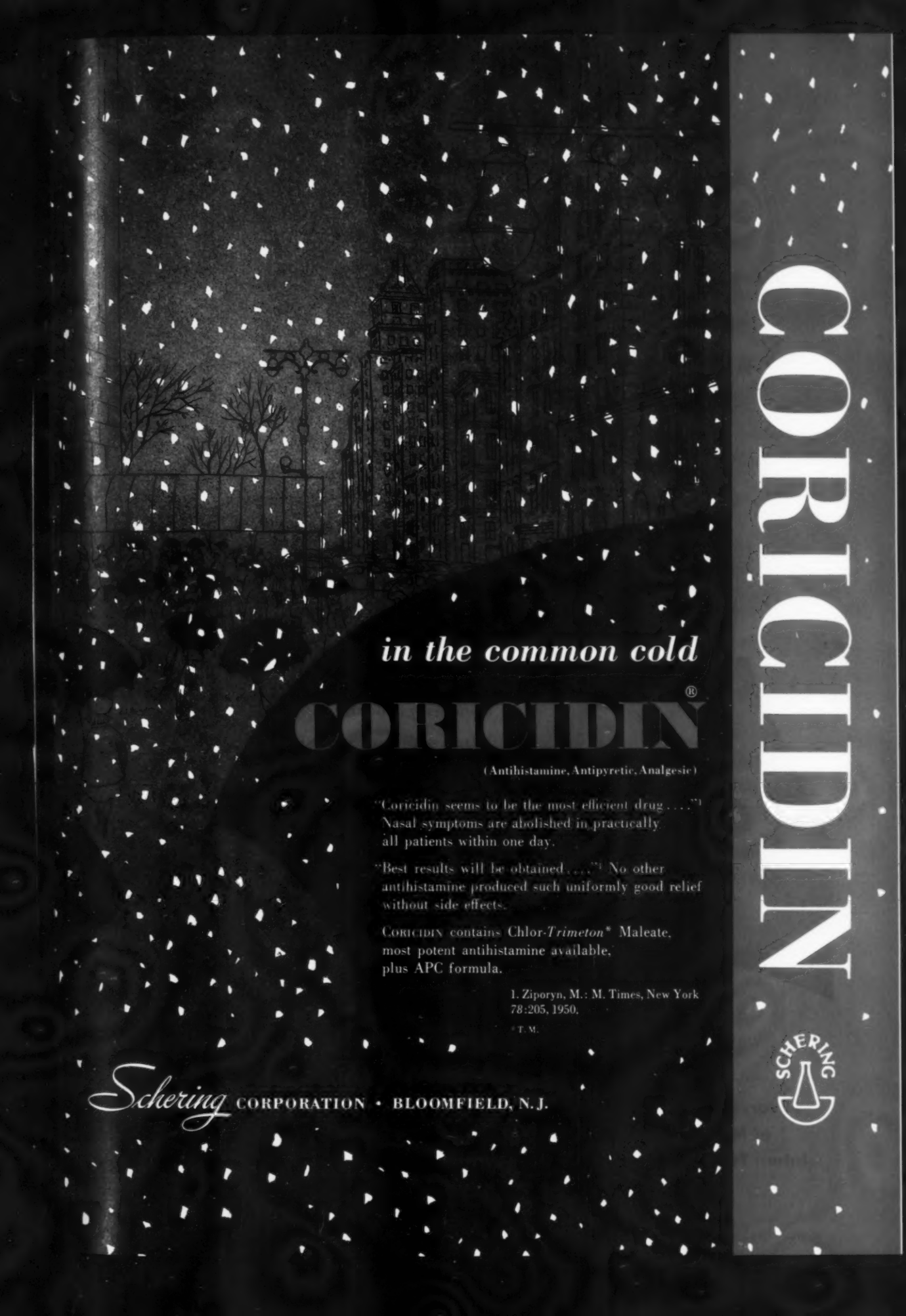

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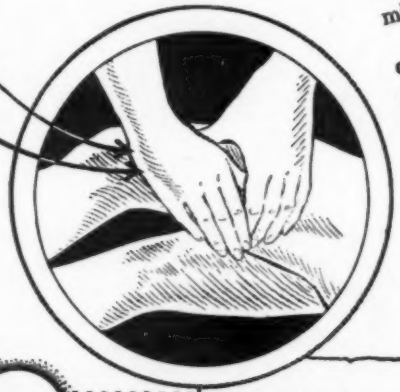

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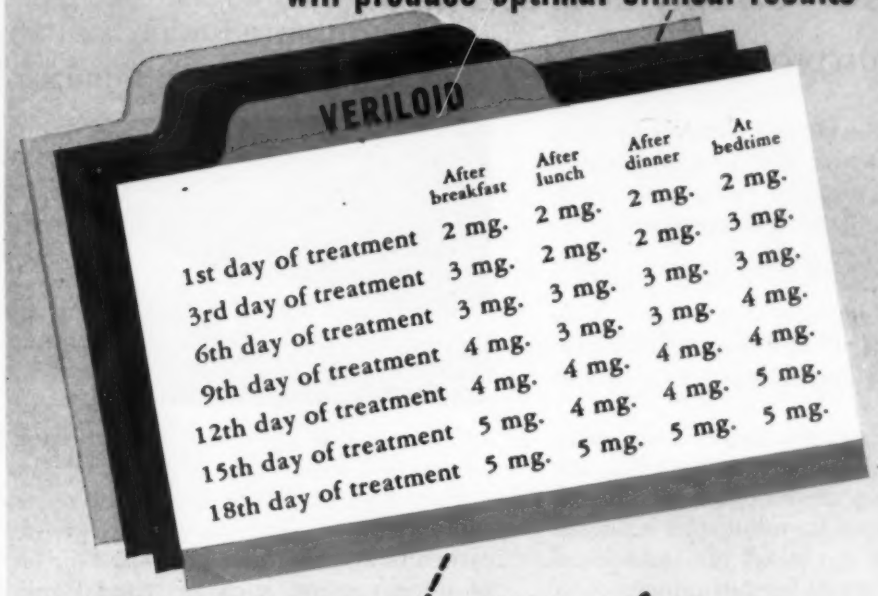
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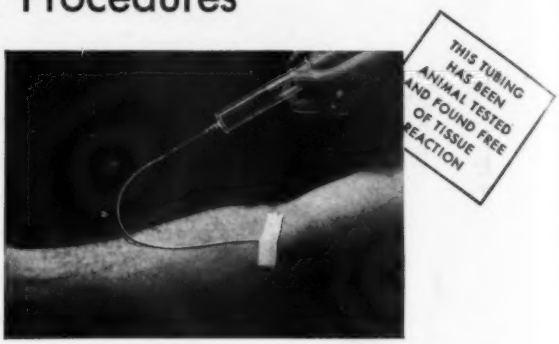

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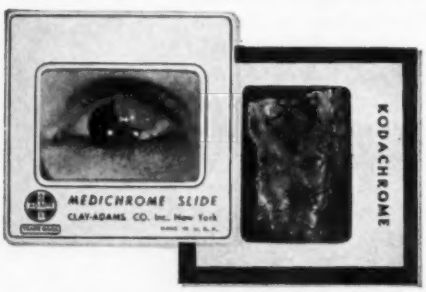

Regardless of your specialty, chances are that Clay-Adams probably has a Medichrome series to cover your field of interest. To mention only a few of the more than forty series available, there are Medichromes covering various aspects of the following specialties: clinical pathology, surgery, dermatology, gynecology, ophthalmology, urology, pediatrics. Subjects are covered from nursing as well as medical phases.

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Detailed descriptions on the following may be obtained from Clay-Adams on request by number:

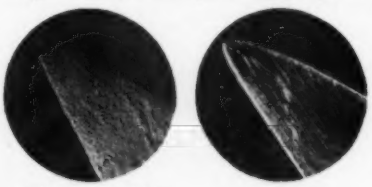
Rust Inhibiting Germicide	(503)
Polyethylene Tubing & Accessories	(447 B)
Kahn Trigger Cannula Outfit	(466)
Medichromes	(339 ML)

Newsletter

FOR THE BIOLOGICAL AND MEDICAL SCIENCES

New Rust Inhibiting Germicide Resists Rust, Film Formation on Instruments and Appliances

RIG concentrate, a new rust inhibiting germicide for the cold disinfection of medical instruments and appliances, protects costly instruments from rust or film even after six months' immersion in RIG, tests reveal. Photomicrographs below show the pitting and loss of cutting edge of a scalpel left in a germicide without rust inhibiting features *(left)*, and absence of such damage from a scalpel left in RIG *(right)*.



Ordinary Germicide RIG

Concentrated in 10 ml. ampules, each ampule of RIG diluted with soft *or hard* water, makes one quart of highly potent germicide. Germicidal and rust inhibiting features of RIG are retained until solution is discarded. RIG is also available in pint cans for hospital use.

RIG is colorless, odorless and stable, and contains no mercury, phenol or formalin. RIG may be safely used on metal, rubber, plastic or glass instruments and appliances.

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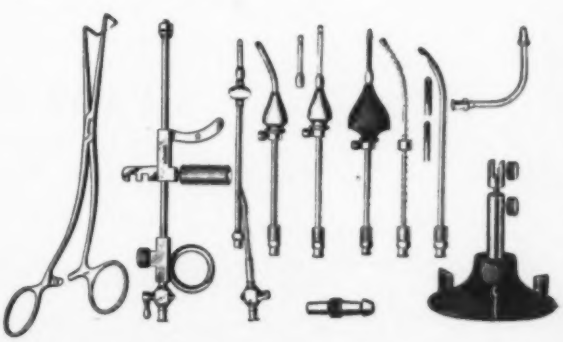

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Neustaedter, T.: Am. J. Obst. & Gynec. 46:530 (Oct.) 1943.

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Glass, S. J., and Rosenblum, G.: J. Clin. Endocrinol. 3:95 (Feb.) 1943

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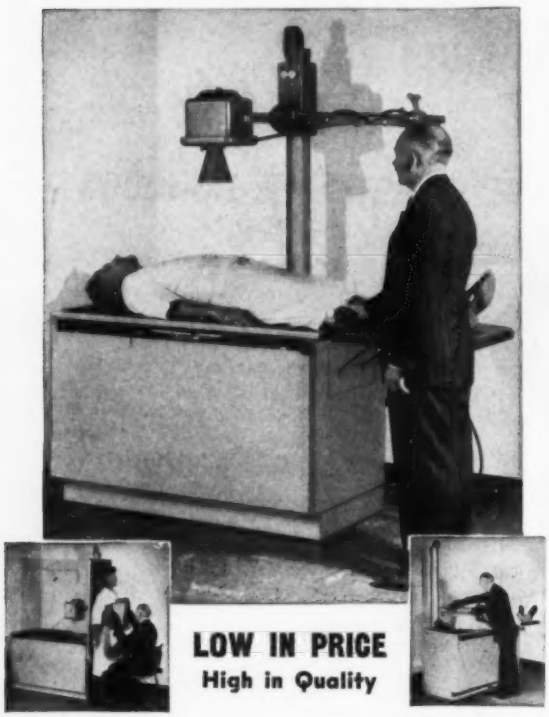

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BIBLIOGRAPHY

Joslin, E. P., et. al: Treatment of Diabetes Mellitus —8 Ed., Phila., Lea & Febiger, 1946—P.241, 247.

Lowsley, O. S. & Kirwin, T. J.: Clinical Urology —Vol. 1, 2 Ed., Balt., Williams & Wilkins, 1944 —P. 31.

Duncan, G. G.: Diseases of Metabolism—2 Ed., Phila., W. B. Saunders Co., 1947—P. 735, 736, 737.

Stanley, Phyllis: The American Journal of Medical Technology—Vol. 6, No. 6, Nov., 1940 and Vol. 9, No. 1, Jan., 1943.

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References

1. Doan, C.A. and Wright, C.S., The Anemic States: Their Causes and Treatment, *Med. Clin. N. Amer.*, (March) 1949, p. 541.

2. Kracke, R.R., *Diseases of the Blood and Atlas of Hematology*, J. P. Lippincott Co., Phila., 2nd ed., 1941, p. 202.

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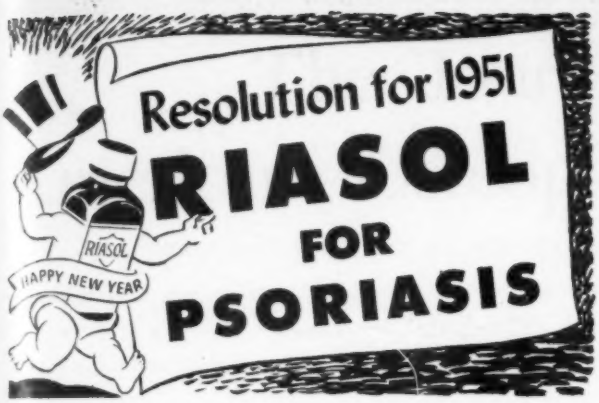

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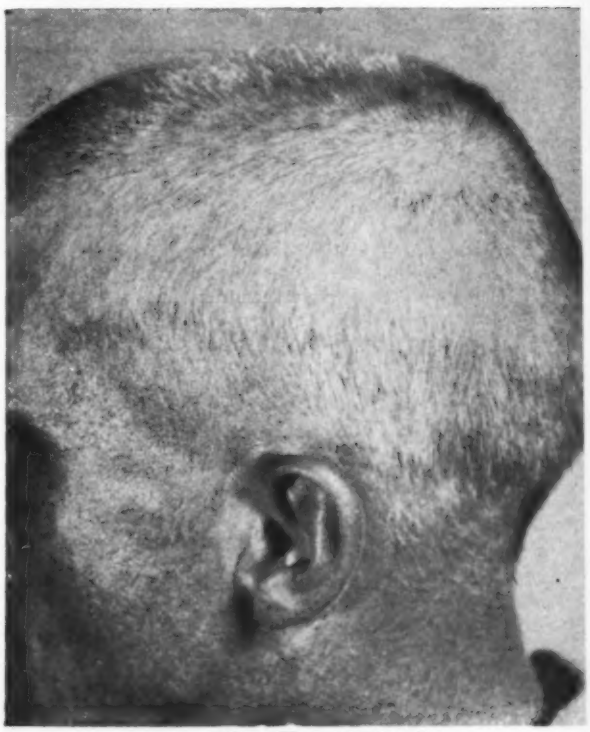

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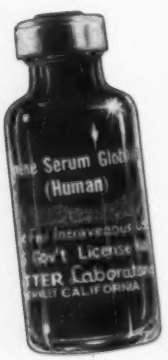

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Lobar Pneumonia and "The Segmental Plane"*

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A review of "what happens" in lobar pneumonia under structural osteopathic care is indeed a review of the basic sciences themselves. Though the application of osteopathic manipulative measures is a matter of physics, which is more easily demonstrated than described, the motivating concept is Still's postulate that anatomy governs physiology and the corollary that anatomy is in turn modified by physiology.

In the expansion of Still's structure-function concept a philosophy of body dynamics has been developed within the osteopathic profession which has long been established clinically and which is being upheld by the most recent physiologic thought. The vasomotor nervous mechanism through which osteopathic manipulative measures achieve much of their results are being restudied and reevaluated so that the sick individual is gaining belated recognition as a major entity in disease.

To the osteopathic physician anatomy is not a corpse. It is a living, feeling, adapting, dynamic, metabolic thing, the integrations of which are dependent upon physical laws. Thus, for example, it is difficult indeed to *know* the anatomy of the pulmonary lobule without understanding something of the biologic principles responsible for its development or the physiologic laws upon which its function depends.

GENERAL APPROACH

Let us go to the bedside of a patient who has recently acquired lobar pneumonia and observe a few of his more obvious symptoms. He has a fever. His breathing is rapid and shallow. He has an unproductive cough. His present condition has developed suddenly and was preceded by a chill. Although he is bedfast he is obviously hard at work for an elevated metabolic state prevails.

Though we must evaluate the patient in terms of his total economy, for it has been mobilized for the purpose of survival, our immediate aim is to gain for the patient an added advantage over the disease. In doing so we should like to consider:

1. The structure-function of the pulmonary lobule in health and in lobar pneumonia
2. The structure-function of the segmental reflex arcs
3. A concept of structure-function of a thoracic spinal segment as a unit of an organ itself
4. The vagal reflexes which produce some of the characteristic protective symptoms
5. Humoral protective responses (respiratory, vasomotor) mediated through the bulb and hypothalamus
6. A philosophy of structural technic.

CONCEPT OF "THE SEGMENTAL PLANE"

As the subject is developed I hope there will be more clearly seen the significance of a perverted structure-function of the segmental reflex arc as it may relate to the pulmonary lobule, for it is my belief that it is through the segmental nervous channels (which are essentially sympathetic) that the process may be initiated and maintained and through the vagal and humoral channels that the protective responses are elicited. It may then perhaps be more easily appreciated that these protective responses are but effects which under ordinary circumstances should not be modified as an end in itself. Thus it is known that treatment of cough, manipulative or otherwise, is not usually in itself an end. Nor is modifying the fever as such to be desired under ordinary circumstances, for to do so is to modify a vital protective effect, thereby adversely altering the body dynamics. As a rule, physiologic relief of symptoms in acute visceral disease is, I believe, achieved through altering the physiology at the segmental plane. It may be accomplished through penicillin, poultices, lymph drainage, or structural adjustment, for example, or a combination of these, but it is accomplished, as I shall try to show, through effecting useful adaptations within the segmental, or horizontal, plane.

PULMONARY LOBULE IN RELATION TO THE "SEGMENTAL" PLANE

The pulmonary lobule is the anatomic-physiologic unit of the lung. Its chief function is to relieve the blood within its alveolar walls of the carbon dioxide it contains and to replace the carbon dioxide with oxygen to be used by the tissue cells. The lobule is roughly cone-shaped having a base which may average the size of a nickel or a dime. At the apex the entering bronchiole subdivides into alveolar branches from which alveolar sacs with their terminal alveoli extend. In addition to the bronchiole there are seven

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Fig. 1. What goes into and out of a pulmonary lobule.

other structures which should be looked at individually, with particular attention to the nerves. These structures are the bronchial artery, bronchial vein, pulmonary artery, pulmonary vein, lymphatics, and branches of the vagus and sympathetic nerves (Fig. 1). The alveolus is the filter itself.

The right bronchial artery carries fresh blood from the thoracic aorta and the left from an intercostal artery for nourishment of the walls of the pulmonary vessels and lymph glands—in fact for all of the pulmonary lobule except the alveoli themselves. The blood is returned in part by way of the bronchial veins down the bronchial tree into the azygos system of veins.

Each pulmonary artery conveys systemic venous blood from the left ventricle for aeration within the alveolar capillary bed where it mixes with blood from the terminal bronchial arterioles which is then returned as arterial blood to the right atrium by way of the pulmonary veins.

The lymphatics of the lobule drain the farthest extension of the bronchial tree and converge in a group of nodes which crowd around the tracheobronchial fork. Thence lymph is conveyed by way of the thoracic duct, and on the right a smaller duct, to discharge below the jugular valves into the left and right subclavian veins.

Through both the sympathetic and vagus nerves within the lobule an integrated two-way system of communication with both segmental and suprasegmental centers is constantly maintained. To appreciate the role of these complementary systems in health and in pneumonia the mechanism of a common reflex arc should be reviewed. We are all familiar with the simple diagram of two neurons synapsing within the cord, but, with the exception of the myotatic reflex, in the human body a three-neuron mechanism is perhaps the simplest that may be found. It consists of the afferent neuron from the skin, soma, or viscera, to the dorsal horn; a connector or association neuron; and the efferent neuron which completes the arc.

ROLE OF CONNECTOR NEURONS

So important are the connector neurons that they constitute by far the greater part of the spinal cord. Somatic connector neurons remain within the spinal cord to form the fasciculi proprii. All sympathetic connector neurons are thought to leave the cord via the ventral root to arborize with their true motor components in either the lateral chain ganglia or the prevertebral plexuses of nerves (Fig. 2). They are able to connect one part with almost any other part (convergence and divergence) if the stimulus or combination of stimuli is strong enough or often enough, or both. They habitually follow certain patterns, whose paths of least resistance are more easily evoked (facilitation).

Within the sympathetic system each preganglionic fiber which leaves the cord may synapse within several ganglia with as many as seventeen postganglionic neurons.[1] This provides the pathway for a diffuse response within the viscera—let us say the suprarenals or the liver as examples in acute infection of the lung or perhaps in reciprocal inhibition of the vessels of the gut.

Nervi vasorum† are found within the pulmonary lobule attending the smallest arterioles, and even the

†Nervi vasorum—both vasomotor and vasosensory fibers.

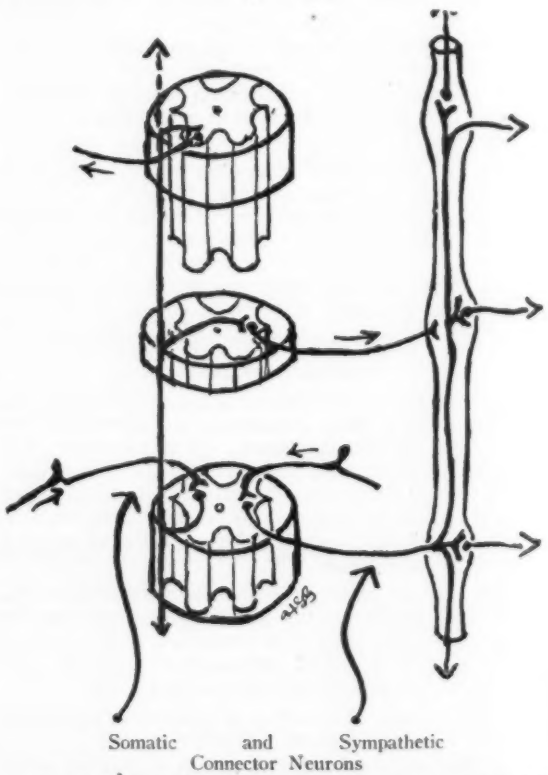

Fig. 2. Note that somatic connector neurons remain within the central nervous system (cord) while the sympathetic connector neurons leave the cord to arborize in the various sympathetic ganglia.

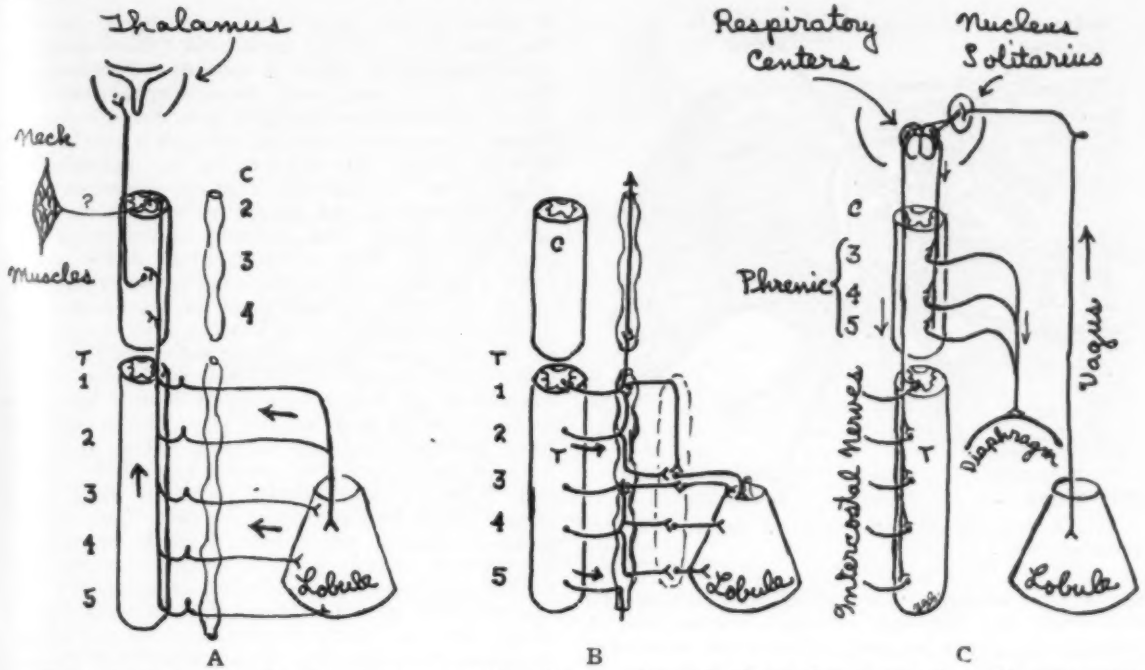

Fig. 3. A—Sympathetic afferent connections. B—Vasomotor connections. C—Vagus afferent connections with motor nerves of respiration.

alveolar capillaries are thought to be so supplied. It has long been held, and only recently demonstrated, that vasosensory reflexes have their afferent receptors within the vascular muscular wall,[2] so we may assume that such receptors are also present in the blood vessels of the lungs. The nervi vasorum entwine themselves around the vessels where they course from their finest branches down to the hilus of each lung. From there, after helping form the pulmonary plexuses, they extend in the posterior mediastinum to join the upper five or six thoracic ganglia of the paravertebral sympathetic chains, mainly, however, at the level of the second, third, and fourth thoracic vertebrae.

We may trace the afferent fibers from their lobular origins directly through the sympathetic ganglia, through the upper five or six white rami where they pass into the dorsal roots to enter their cell station in the sensory ganglia. From there the cells extend themselves into the corresponding segments of the cord to course upward in the white matter, mainly to the level of the third and fourth cervical segments (Fig. 3A), where they arborize with many connector neurons which in turn arborize with the sympathetic and perhaps with the somatic motor nerves to the skin and striated muscles of the neck. In tracing the efferents from the lobule to the cord, we find them traveling a similar path, but for ganglionic synaptic interruptions, and entering the spinal trunks from the first to sixth thoracic white rami, where they travel along the ventral roots to enter the horn wherein their cell bodies reside (Fig. 3B).

Although Grinker and Bucy[3] state that the third and fourth cervical segments are areas of referred pain from the lungs and Pottenger[4] demonstrates the referred trophic changes in the cervical area, osteopathic knowledge has led us regularly to find associated upper thoracic osteopathic lesions, especially third and fourth thoracic lesions. The mechanism seems to be as follows: A group of previously induced upper thoracic spinal lesions may initiate lung pathology by producing synaptic alterations within the cord (facilitation). The reflex arc through which the spinal lesion is initiated is from the osteopathic lesion through the segmental afferent neuron to the cord, the segmental connector neuron within it, and the segmental efferent motor neuron back to the spinal lesion, which completes the arc. The reflex arc through which the spinal lesion is *maintained* would, I think, be a proprioceptive postural reflex which would possibly travel along a similar arc. Its maintenance, therefore, would not be dependent upon pathology within the lung but would be sufficient unto itself. (See sections titled "The Spinal Segment and Luschka's Nerve" and "The 'Jumpy' Synapse.") The heightened stream of impulses through the involved cord segments results in vasomotor overtones which may bear upon the lobule and "weaken" it through the vasomotors of the vessels which supply the lobule.

It can be seen, however, that the referred effects of lung disease would not in themselves be manifest solely within the upper thoracic spine, for it is through the afferent terminals, whose synapses are found within the cervical cord, that certain cervical reflexes are maintained (Fig. 3A). The osteopathic physician, however, correctly centers his manipulative therapeutic endeavors upon the segments which helped predispose the lobule to disease, but treats all other lesions wherever found. In centering his attention upon the third and fourth thoracic segments and those joints contiguously involved he is attempting to raise the threshold of the original facilitated reflex arc which determined the site of the acute disease, by improving the nourishment of those neurons whose cell bodies live within the spinal cord on that segmental plane.

As a result of the edema of the alveolar membrane and the altered pH of its fluids in early pneu-

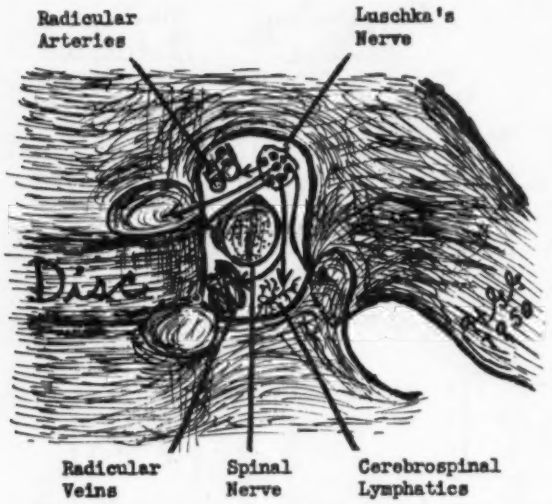

Fig. 4. Structures which transverse the intervertebral canal (diagrammatic).

monia, noxious stimuli are transmitted to the cervical cord (Fig. 3A). Through connector neurons viscero-visceral reflexes are initiated, which would reasonably involve the heart via the three cervical cardiac nerves and the diaphragm via synapses with the phrenics (Fig. 3C) and involve vasomotor adaptation of other organs in other planes (because of their more diffuse ganglionic synaptic response). See Figure 2. Through connector neurons viscerosomatic reflexes are mediated which eventuate in changes within deep somatic tissues of the neck which are manifest as reflex lesions of the cervical spine.

THE SPINAL SEGMENT AND LUSCHKA'S NERVE

The spinal column should be regarded as more than a system of levers involving adaptation to gravitational and respiratory forces, which incidentally protects the spinal cord. Not only does the spinal column protect the spinal cord but may be considered to nourish it, for the nourishment of the cord is dependent largely upon the integrity of the spinal joints. Through the foramina pass not only the emerging spinal nerves, but the arteries which nourish and the veins which drain the cord, its roots, its mantles, the periosteum, the spinal ligaments, the joint tissues which help make up the foramina, and parts of the adjoining vertebrae themselves[5] (Fig. 4).

In addition to the systems mentioned which traverse the intervertebral canal there are the lymphatics which drain the cord and its membranes,‡ and the trunk and branches of the so-called sinu vertebral or recurrent nerve (Fig. 5), first described by Luschka.[5] This small nerve trunk "enters the vertebral canal and distributes its filaments to the vertebrae, to the venous sinuses, to the arterial branches, to the dural sheaths and the extradural fibroadipose mass to the ligaments, etc."[5] This is part of an intricate description given by Guy in his series, "Vertebral Mechanics," which first appeared in THE JOURNAL OF THE AMERICAN OSTEOPATHIC ASSOCIATION in 1930 and was reprinted in the *Academy of Applied Osteopathy Year Book*, 1949. It seems to have gone quite unrecognized clinically for what it is, both by members of the osteopathic profession and by others in spite of the obvious importance which was clearly focused upon it by Guy. Much searching has produced little further description of it in current writings§ although it may be quite as Guy[5] stated, "the keystone of the understanding of *the lesion*." It typically originates from two roots, one cerebrospinal and the other sympathetic. The sympathetic comes from a ramus of the sympathetic chain and the other, which is often double, comes from a spinal nerve to penetrate the operculum and supply the vertebral canal and all that it contains. This is the sinuvertebral or Luschka's nerve. (Fig. 5). In the thoracic region Guy observed, the cerebrospinal root arises near the internal border of the costotransverse ligament, a costal branch leaving it to traverse the neck of the rib. The sympathetic roots, he stated, are quite variable. They, of course, follow with the extensive ramifications of the vessels, and, it would be concluded, continue along those vessels which feed and drain the cord. The cerebrospinal component which may, I think, be somatic-sensory (or mixed) joins it along its course, and from there on the two cannot be told apart. We have only to reflect upon the significance of the distribution of this nerve on all the structures within and without the spinal canal, including the nourishment of the cord itself, to envision what far-reaching consequences the osteopathic lesion may entail.

VASOMOTORS OF SPINAL CORD

Recent studies have suggested that of the thirty-one segmental arteries which feed the spinal motor and sensory roots only five to eight on either side[1] join the spinal arteries to supply the nerve cells of the cord. (The veins are likewise said to be inconsistent though they more frequently anastomose with the veins of the spinal cord. Though there is as yet no way to tell through which foramina the vessels may make anastomoses with the vessels of the cord. The thoracic segments are said to have the poorest, and the cervical the richest, segmental blood supply. This irregularity, I think, should not affect the segmental innervation of the cord via Luschka's nerve. Read details of its segmental connections with spinal roots and the sympathetic chain as described by Guy.[5]

The anterior and posterior spinal arteries are formed from the two vertebrals after they course around the atlanto-occipital articular capsules to enter the topmost portion of the spinal canal. These provide the cervical cord with a rich circulation. Four or five radicular arteries (two or three to a side) from lower segments of the neck may join them in their course. In the upper thoracic region perhaps six or eight of various caliber are said to reach the cord (which would be three or four to a side).

The vertebral arteries receive their innervation from the upper thoracic cord centers by way of synapses located mainly within the inferior cervical or stellate ganglia. Inasmuch as there is claimed to be a scarcity of segmental arteries which reach the upper thoracic segments of the cord, and inasmuch as the

‡It seems evident that the central nervous system has its own lymphatic system, separate and apart from the cerebrospinal fluid, which drains the central nervous system and connects with the general lymphatics. The probable mechanism and its bearing on the immune processes will be considered in another paper which is in preparation.

§Roofe[6] is cited as having found (1940) that each recurrent nerve was distributed not only to the dura, but "to the ligamentous structures in the anterior spinal canal, extending 2 vertebrae below its origin." H. L. Litton referred to it in the "Handbook on Osteopathic Technique" (1941).[7] Korr[8] in a personal communication states, "We have come to believe that [Luschka's nerve] may play an important role in altering circulation through the cord and its membranes, which in turn may be responsible for important secondary and more lasting effects of visceral and somatic disturbances."

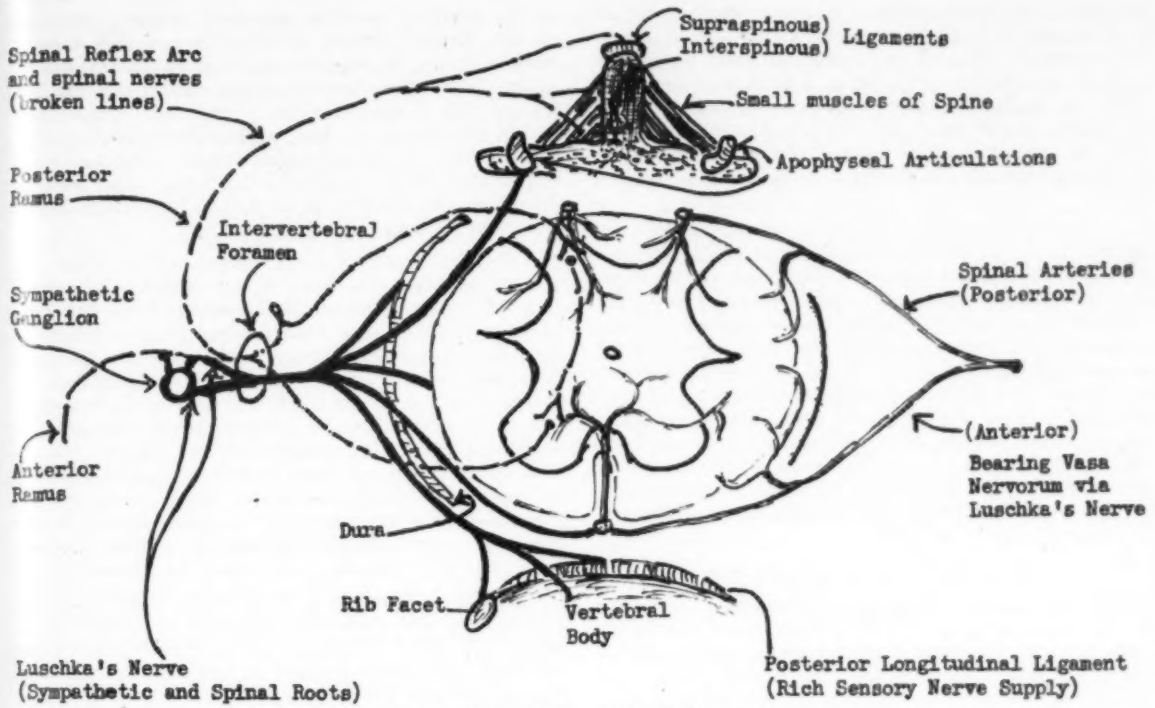

Fig. 5. Distribution of Luschka's nerve.

upper thoracic segments are supplied to some extent indirectly off the vertebrals, it should follow that osteopathic lesions of the cervical spine may, by way of Luschka's nerves, affect adversely the circulation of blood within both the cervical and upper dorsal segments of the cord, for wherever there are arteries or arterioles, vasomotor nerves are found. In our patient with lobar pneumonia, the vasomotor nerves which supply the cord arising from the inferior cervical or stellate ganglia (whose preganglionic cell bodies lie within the upper thoracic segments of the cord) should play a most important nutritional role. Burns[9] has observed microscopically the effects of artificially induced spinal lesions in the cord. She states in effect that in "lesioned" animals the cells of the anterior, posterior, and lateral horns of the segments affected by vertebral lesions show degenerative changes which parallel the sequence within the gray matter of "hyperemia, congestion, edema, minute hemorrhages with coagula, [followed in time by] fibrosis and ischemia."

In experiments of Cole[10] on the effects of artificially induced atlas lesions of 6 months' duration in the rabbit, there were observed minor histological changes in the cervical and first four thoracic segments including congestion of the blood vessels and infiltration of erythrocytes into the tissue spaces of the gray matter, thickening of the media of the arterioles, a slight swelling of the anterior horn cells, and a slight glial proliferation. No changes were noted in the white matter.

THE VAGUS AND THE BULB

In order to have some understanding of the mechanism of some of the protective reflexes which are evoked in pneumonia, let us turn first to the sensory receptors of the vagus and then to certain centers within the midbrain and the bulb. These are being termed "suprasegmental" reflexes whose afferents are via the vagus nerves (Fig. 3C).

Vagal receptors are found imbedded more or less within the substance of the bronchioles, extending into the lobules to reach the alveolar ducts, air sacs, down to (but not including) the alveoli. Therein six varieties of vagus sensory endings have been counted,[2] whose axones are carried to the base of the bronchial tree, thence up the vagi to reach their bodies in the ganglia nodosa where the dendrites pass through the jugular foramina to terminate in the solitary nuclei which are close to the expiratory center of the bulb. They are nonadaptive proprioceptors responding to the stimulus of altered tensions which is generally termed stretch.

Within the bulb and pons resides the respiratory center (Fig. 3C) of which the expiratory and inspiratory centers are a part. The inspiratory center is headquarters for those tracts which descend through the cord to synapse at the level of the fourth and fifth cervical vertebrae with the emerging phrenic nerves. The phrenics course through the thoracic operculum to motivate the dome of the diaphragm in its inspiratory phase. (In deep or difficult inspiration the respiratory centers call the lower intercostals and abdominal muscles into play.)

Crowning this complementary system there is still another group of cells known as the pneumotaxic center which, though normally quiescent, in the presence of lobar pneumonia or other fever is called to the fore.[11] Its strategic value in this emergency seems to be through its direct connections with the hypothalamic heat regulating centers which are in turn in direct "leased wire" communication with the respiratory center and other higher centers of the brain.

The vagal proprioceptors, whose sensitivity is exalted by the edema of the pulmonary lobule,[2] induce the expiratory centers into a more dominant role,

producing the phenomenon of shallow breathing which is pathognomonic of lobar pneumonia. This is a protective reflex, induced apparently to limit the excursion, hence activity, of the lung. The shallow breathing tends to produce a higher carbon dioxide value of the blood, which acts upon the pneumotaxic center to produce an increased respiratory rate. (Shallow breathing may also be induced as a result of pleural pain.)

Another protective reflex response is that of cough. It is activated probably by another type (or types) of stretch receptor mediated through the vagus to reach the conscious level while the patient is awake, but active enough in sleep. In cough the vagus (sensory and motor) hypoglossal, glossopharyngeal, accessory, trigeminal motor, facial, intercostal, phrenic, and spinal and abdominal motor mechanisms all take part and continue to do so until the lobular nonadaptive vagal proprioceptors are relieved or until volitional control supervenes; hence the common use of syrups containing drugs aimed to quiet the proprioceptors by loosening the phlegm and narcotic drugs to depress the higher centers as well.

FEVER AND THE HYPOTHALAMUS

In our patient with early pneumonia the toxins of the pneumococcus which course from the lobule through the vascular hypothalamus are quick to excite the heat regulating centers located there. The heat conservation center is soon called into play, producing: (1) a constriction of the vessels of the skin, (2) constriction of the sweat glands, (3) erection of the body hairs, and (4) a chill. The syndrome of heat conservation would be expected to continue until a level of internal balance (homeostasis) compatible with the dynamics of the disease is effected. It may be modified in the course of the disease to suit the patient's internal needs (as shown on the temperature chart) probably more often without the phenomenon of sensible sweat.

These are some of the mechanisms, as I understand them, whereby the protective machinery of the body is utilized in lobar pneumonia. They are effected via the suprasegmental plane. Because they are not effected by way of the segmental sympathetics one can perhaps more easily understand why under ordinary conditions these mechanisms, as such, are not to be greatly disturbed by the use of drugs which depress the suprasegmental nuclei in the brain.

THE "JUMPY" SYNAPSE

Korr[12] in his scholarly monograph, "The Neural Basis for the Osteopathic Lesion," has given perhaps the best explanation to date of the segment as a working unit in visceral disease. Starting with the spinal lesion and focusing his philosophy upon the cord, he evolved three postulates which I have tried to follow in the preparation of this manuscript. He said in substance that:

1. Overstimulation of sympathetic fibers in the segment of a lesion may initiate visceral disease.

2. The untiring sources of impulses which maintain the osteopathic lesion are proprioceptive in nature.

3. By appropriate manipulative therapy the noxious proprioceptive stimuli may be modified or abolished, thereby relieving the segmentally related viscus of abnormal sympathetic stimuli.

The most significant contribution to osteopathic thinking is the concept of the proprioceptive nerve ends as "the untiring source of impulses" in the maintenance of the lesion. These receptors are found between muscle fibers, in muscle spindles, in joint capsules, in the periosteum beneath tendinous insertions, and within the fascial sheaths. They are also found within the mesentery and, as has been shown, within the structure of the pulmonary lobule. The dura mater is probably richly supplied with them. They respond mainly to the stimulus of stretch. (Perhaps distortion would be a better word.)

It is characteristic of proprioceptors that they are nonadaptive (or very slowly adaptive) in function. By this is meant that, unlike touch or smell, they do not become "silent" but may continue to discharge impulses as long as the stimulus is there. Adaptation in its varying degrees is a dynamic mechanism and is not to be confused with what is termed "fatigue." To enjoy the sense of touch, for instance, one must constantly manipulate the object, for the sensory receptors of touch readily adapt themselves; to enjoy the adaptive sense of smell of a perfume the glass rod from the bottle is wafted briefly before the nose. But a backache will, as a rule, continue until the proprioceptors are eased of strain.

While I have tried to convey Korr's theorem by striking out from the focus of infection in the lobule to widely scattered sites, Korr[12] did so by focusing his attention upon a segment of the cord. He emphasized the principles of facilitation and convergence upon the synapses which bear upon the final common path. Facilitation is conceived as the synapses being "jumpy" or on edge. There is a lower threshold of the synapse to the wave of excitation it receives, so that a lighter volley than normally required may set it off. Or to view it another way, a normal volley may set off many more synapses than usual.

CONVERGENCE AND THE "JUMPY" SYNAPSE

It is well established regarding convergence that fibers from many sources converge upon every motor or connector neuron of every segmental plane. From the cerebral cortex, from the reticular formation (wherein lie so many vital centers), from the vestibular nuclei and those of other special senses, from centers which are relay stations from the cerebellum, cortex, thalamus, and hypothalamus (perhaps in part directly from the latter), from sensory areas up and down the cord, and from sensory nerve ends anywhere within the body, we can picture fibers converging upon a facilitated "jumpy" synapse of the segmental plane. Noise, drafts, too much company, osteopathic lesions, fear of death, pain, aspirin, diabetes, nephritis —the sum of our reactions to internal and external environment converges upon the weakened synapse, as it does upon all other synapses which determine the final common path, and finds an easy outlet through it to the diseased segmental plane.

It follows then that any measures taken which will decrease the degree or varieties of adventitious stimuli which converge upon the "jumpy" synapses should aid, to the extent of the decrease, in recovery from disease. Hence the physician advises rest, fluids, quiet; gains the patient's confidence; supports normal body chemistry and excretion; tries to relieve pathology, osteopathic or otherwise, not only within the segmental plane but wherever found.

Some of these measures of course are instituted by all physicians, whatever their school, for it amounts

to doing what comes naturally. It should be remembered, however, that these are all indirect or supportive measures (except to the extent that they directly affect the ailing segment) which are instituted to protect the "jumpy" synapse, thereby shielding from further insult the injured segmental plane.

In the hypothetical patient under consideration it is conceived that the pneumonic process within the lobule has aggravated a plane or planes of facilitation within the cord which may have been subliminal as a result of segmental spinal lesions, but which now involves a group of "jumpy" synapses which opens otherwise silent somatic and sympathetic motor paths. Those somatic pathways are probably by way of regular motor neurons which make muscles contract. The majority of impulses, however, are received by the muscles and their attachments, involved in the preexisting segmental spinal lesion, for their receptors have been previously subliminally (subclinically) excited by the strains they have had to bear.

Those muscles which I feel are most responsible for the maintenance of the subclinical lesion, and which are now receiving an added volley of stimuli, are those small deep muscles which lie nearest the spine (Fig. 5): the rotatores, interspinales, intertransversales, and, to a lesser extent, the multifidi, and semispinales (and their modifications in the suboccipput). It is these small muscles which lend themselves to minute adaptations of posture and which, therefore, must be prepared to report those changes to the cerebellum and thalamus. It is these small muscles, as individuals, which would take the greatest beating under strain.

To an even greater extent, I think, it is within the fascia which surrounds the joint, within the capsular and articular ligaments, and within tendinous insertions of the muscles that the most sensitive and least adaptive proprioceptors are to be found.

A PHILOSOPHY OF STRUCTURAL TECHNIC

The emphasis upon physiologic measures in the treatment of disease, which (we are prone to think) is the distinguishing feature of the osteopathic profession, seems to vary widely in its technical application even among the more skilled osteopathic practitioners. Thus Wilson,[13] in reviewing the work of a small number of osteopathic physicians recognized as being experts in manipulative therapy, was struck by the diversity of gross technics which were employed. He concluded that, with one exception, all methods had one thing in common—an increase of drainage of the lymph. (I feel, however, that all secured lymphatic drainage, for one cannot administer an adequate osteopathic manipulative treatment without effecting some increase in drainage of the lymph.)

It is evident that in osteopathic lesion therapy, whether in pneumonia or other conditions, sound approach is based upon the tissue feel. Thus, according to Wilson,[13] Charles Medaris, with one hand on the heads of the ribs, abducts the arm and employs it as a handle of a pump. George Riley tells how he patiently, persistently gave all he had to relaxing excruciatingly tender spinal muscles until he secured *"that feel."* Harry Gamble springs the spine and elevates and separates the ribs gently but firmly as *prudence permits.* O. M. Walker relaxes all deep muscles, getting as much motion in each vertebral joint as possible. Ralph M. Crane lifts the third and fourth thoracic segment until he *senses* a relaxation of the spine. John A. MacDonald had the objective of maintaining maximum diaphragmatic excursion by grasping the lower ribs and increasing their excursion with the respiratory rhythm. A. T. Still treated pneumonia by correcting the ribs at their spinal articulations. Wilson himself advises "slow rhythmic traction until the occiput is *felt to lift*" and extension of the third and fourth thoracic vertebrae (as outlined by Crane) *"until the patient lets go."*

The one thing most evident in those remarks is that the physician goes along with the patient until he senses the give of the tissues. Even MacDonald went along with the patient in securing rhythmic costovertebral motion of all the thoracic joints by using the lower ribs as levers. The good results of every technic employed are, I believe, achieved to a very large extent by modifying those impulses to the proprioceptor endings within the small deep muscles, the tendons, ligaments, and articular capsules of those somatic areas of greatest segmental strain. Holding until one senses the tissues give (the feel of the tissues), gently springing the spine, stretching the deep muscles—all are measures which I think are directed toward the nerve endings whose sense of altered tensions never tires in the job of informing the physician of the segments wherein the patient's trouble lies.

The feel of the tissues by the physician, I hold, is mediated little through the sense of touch, or heat, or cold, or pain,‖ but by receptors similar to a type found plentifully within the muscles, joints, and tendons themselves. They are the pacinian corpuscles resting within the deeper layers of the skin of the fingers, which mediate the pressure sense. It is evident that they do not tire, for constant contact is needed for evaluating the responses we are engaged in eliciting. Thus we sense by "proprioception" the feel of the tissues; we "live with the patient," so to speak.

PHYSIOLOGICAL RESPONSE TO OSTEOPATHIC MANIPULATIVE TREATMENT

As a result of structural treatment in our patient with pneumonia, those sources maintaining pathology through reflex facilitated paths have been, we like to think, in some measure and for some time removed so that fresh blood may flow through some of the capillaries of the lobule in a less embarrassed way. There has been improvement for a while in vasomotor tone. The heart has been relieved a bit of its embarrassing load. With evidence of perspiration, the temperature may drop somewhat. The patient breathes more easily, and may drop off to sleep. He has gained a measure of advantage over the disease. This advantage, I should like to repeat, has in my opinion been achieved through altering the structure-function within the segmental plane; the modifying of the obvious effects, the symptoms, would not have been attempted as an end in itself. Aspirin and phenobarbital (or phenobarbital alone) could have modified many of the outward manifestations, but the results would have been secured by depressing certain suprasegmental centers and thereby depressing natural protective responses.

In directing structural technic toward modifying the vasomotors in the patient's segmental plane, it is reasonable to suppose that the lymphatic drainage of

‖The use of all the senses (except perhaps that of taste) are of course needed in order to make a full diagnosis. H. V. Hoover[14] has described methods of detecting the changes in temperature and texture of the skin and muscles found in different types of spinal lesions.

the spinal tissues has been aided as well as the drainage within the lobule itself, and that there has been effected a surge in the specific antibody response initiated through release of fluids from that segmental plane, which acted in turn upon the process within the diseased lung. The vagal stretch receptors within the lobule walls have likewise been eased of strain, accounting for an easier, deeper respiration and perhaps a lessened tendency to cough. The synapses within the cord have become less jumpy, we may opine, and some of their pathways have been closed. As to the vasomotor fibers of the cord, we may assume that their constrictor tone has been improved and that a fresher blood flows through the spinal segment, thereby relieving it of its edema and checking in some measure the pathologic trend within the motor or connector cells.

What then of the segment as a unit—what of the distribution of Luschka's nerve (Fig. 5)? That measure of relief of the proprioceptive barrage throughout the segment, I think, would be enjoyed by all the tissues served by Luschka's nerve. The cord should work more capably; the blood should flow through the foramina more abundantly, thereby nourishing and draining all those tissues within the vertebral canal. There should be then less meningeal swelling and more drainage of the lymph, hence less proprioception within the spinal ligaments and joints which Luschka's nerve supplies. We should then expect more nourishment to the marrow of the involved vertebrae and ribs with the consequence that more blood may then be formed. Thus the imaginative¶ physician may sense by the *feel of the tissues* the physiology he is modifying in securing greater motion in the joint.

RELATED PHYSIOLOGIC FACTORS

In developing this subject I have found wide areas of vasomotor knowledge which are today still unexplored, that I have tried by inference to map. I have not dwelt upon the role of the other large intrathoracic viscus, the heart, which is severely taxed in pneumonia, nor upon the suprarenals, kidneys, thyroid, pituitary, or spleen which fulfill heroic roles. I have purposely avoided the vasomotor roles of the carotid and aortic sinuses and bodies which take over special functions when the going gets rough.

In this paper I have omitted discussion of the relationship of the rib heads to the sympathetic chain, which Burns[9] has found to participate directly in the vertebral lesion complex.# I have not gone at length into the physiology of the lymphatic system or the relationship of those immune processes which it aids. I have only mentioned the phenomenon of inhibition, which is mutually synergistic to facilitation, and plays a dynamic, positive role. I have not been able to evaluate the significance of the irregular blood supply to the spinal cord (if such is actually the case).

THEORETIC CRANIAL APPLICATION

Finally, I have not discussed the claimed specific role of craniosacral osteopathic manipulative therapy for the reason that the mechanism is unknown. Whether the mechanism is neural-supportive, via the parasympathetic system downward; humoral-supportive, through modifying the vital centers locked within the skull; via cerebrospinal fluid fluctuations;[15] or through some combination of these directed through humoral and nervous media to the segmental plane has not as yet been told.

The skull bones act (and are acted upon) as levers, as are the bones which make the spine. Though I have found no mention of the type of sensory receptors within the cranial dural membranes, I think it may safely be said that they are proprioceptive, tirelessly responding to the stimulus of stretch. Impulses from the cranial dura are carried via the fifth, seventh, ninth, and tenth cranial nerves to synapse mainly within centers in the bulb. I think it is significant osteopathically that afferent fibers from the cranial dura pass into the cervical cord by way of the twelfth cranial nerve.

Rollin Becker[16] in treating pneumonia in terms of the Still-Sutherland[15] philosophy, has made the intriguing observation that to the extent that the thoracic cage is locked in this disease, so are the cranial base and vault. In a small number of cases treated the lesions usually found have been a relative restriction of the occipitomastoid and petrobasilar articulations. Becker claims that a characteristic "toxic" feel of tissues is found. In pneumonia involving one lung only, Becker finds the lesions are on the homolateral side, which would suggest to me the essential sympathetic "segmental" nature of the process by way of the cervical sympathetic chain. Their presence may conceivably have a profound effect upon the whole organism through mechanisms which are best appreciated by those who have studied the cranial concept.

In closing I should like to venture that in the application of the Still-Sutherland philosophy there seems to coincide an integration of the physician's delicate proprioceptive sense with the subject's facilitated proprioceptive sense, which represents the feel of the tissues in the highest known degree. I would say that it is largely through Sutherland and his followers that this rare faculty of healing is being developed and contained within our ranks.

SUGGESTED GROSS RESEARCH

Some anatomical relationships could be systematically checked by the departments of anatomy in the osteopathic colleges which might help in applying the osteopathic concept to diseases of the lungs as well as to other diseases. I suggest the following:

1. From cadavers, gather statistics regarding the levels at which the left bronchial artery leaves the intercostal artery (because of the bronchial artery's nutritional role in the lung and its intimate relationship with the costovertebral joint, thereby possibly affecting the vasomotor supply to the tissues of the lung through edema of the paravertebral ganglia).[9]

2. Record the levels at which, and the frequency with which, the spinal root arteries reach the spinal cord in a given number of cases (because of their functional significance in vasomotor reflexes to the cord).

3. Record the levels at which, and the frequency with which, the spinal root veins anastomose with veins of the spinal cord (for the reason given above).

4. Examine to see if any sympathetic fibers leave the superior cervical ganglia to innervate the vertebral arteries (because of possible significance in the atlanto-occipital articular lesion).

5. Carefully study and report on the connections and ramifications of Luschka's nerve (because of its apparent importance as a source of referred vasomotor pathology to the cord and its containing spinal segment as a result of postural strain).

¶Imagine—to bring before the mind's eye; to conceive in thought. The Consolidated-Webster Comprehensive Encyclopedic Dictionary. Book Production Industries, Chicago, 1948.

#This subject has been briefly considered in an article prepared for publication in the *Academy of Applied Osteopathy Year Book*, 1951.

SUMMARY

In acute infectious visceral disease there are postulated to be:

1. A sympathetic "horizontal" plane of reflex phenomena which is the plane of the primary disease (I have called it the segmental plane.)

2. A "vertical" plane, the reflexes of which are initiated within the viscus, which elicits integrated protective responses through the higher centers to produce many of the classic symptoms of the disease (I have termed it the suprasegmental plane.)

3. A general supportive metabolic response (normally) which is elicited via humoral channels within higher (chemoreceptor) centers of the brain

4. Facilitation within the spinal cord of the involved segmental plane as an integral part of the pathologic process

5. Facilitation of the cord segmentally is maintained by way of Luschka's nerve through the nervi vasorum it carries.

It is further postulated that:

6. Any measure directed toward alleviating the pathologic process of the segmental plane is physiologic.

7. Osteopathic structural therapy achieves its beneficial effects primarily through physiologically modifying vasomotor tonus throughout the segmental plane.

8. The vasomotor tonus is modified through alleviating adventitious proprioceptive barrages initiated or maintained within the segmental plane.

9. Those barrages originate mainly within joint capsules, ligaments, tendinous insertions, and fascial sheaths, and between muscle spindles of the small muscles and larger muscles of the vertebral column, and are probably of physiologic importance in that order.

10. Barring direct insult and developmental defects the osteopathic spinal lesion precedes the acute visceral lesion and in turn may be reinforced by the acute visceral lesion.

11. In lobar pneumonia the osteopathic spinal lesions are found typically in the third and fourth thoracic and one or two contiguous vertebrae above and below.

12. There are frequently associated osteopathic lesions in the cervical spine.

13. The application of osteopathic structural therapy in pneumonia effects a proprioceptive release which is anabolic in character throughout the organism.

14. This release is best achieved through the faculty of proprioception as applied to osteopathic structural technic.

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Differential Diagnosis of Hemolytic Disease and Hepatic Insufficiency*

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Sir Thomas Lewis[1] said "The continued separation of disease into types can be pressed too far, and a subdivision which may perhaps have some immediate practical use can help to conceal underlying mechanisms held in common and thus to hinder progress in studying disease." The longer one is confronted with chronic disease, the more he is persuaded that many diseases are not sharply defined entities but rather transitions of morbid states, one to another. It is true that during these transitional stages therapeutic approaches may vary, and only by understanding the natural history and pathogenesis is it possible to apply intelligently a particular indicated plan of treatment. So it is in dealing with hemolytic disease and hepatic insufficiency. On the surface it would appear that there should be little difficulty with these conditions, but we have been plagued with classifications and innumerable tests of liver function which, instead of clarifying the situation, have, in actual practice, made the picture more confusing.

Icterus is not a consistent finding in either hemolytic or hepatic disease, and yet, fortunately, it has served as the single reminder that these two conditions often coexist and that not infrequently they merge. It is a well-known fact that the prolonged hyperbilirubinemia of hemolytic disease by the resulting anoxia and mere deposition of pigment in the liver will result in parenchymal cell damage and hepatic insufficiency. Voit,[2] citing the work of Kalk, in addition to his own observations, has shown that not infrequently is there a transition of chronic epidemic hepatitis into hemolytic icterus with increased fragility of erythrocytes, et cetera. This presentation will deal with the mechanisms and pathogenesis of jaundice and will of necessity

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include a simplified discussion of tests of liver function relative to their importance in differential diagnosis.

The liver is the largest glandular structure in the body, the functional unit of which is the liver lobule. The lobules are held together by the connective tissue of the peripheral spaces with their network of blood vessels, nerves, and lymphatics. All the functions of the liver represent the sum total of physiological processes taking place in these individual parts. The liver lobule thus bears the same relation to the whole organ as the nephron does to the kidney.

Three basic structures enter into the formation of a lobule: parenchymal liver cells, bile canaliculi, and blood vessels. The liver cells are arranged within each lobule in cords that radiate like the spokes of a wheel, from the center which contains a central vein. All the central veins empty into sublobular veins which come together and form the main tributaries of the hepatic vein. While on one side the liver cell trabeculae are separated by venous sinusoids converging toward the central vein, on the other side there are found coursing between the trabeculae the bile canaliculi into which the constituents of the bile are emptied by the hepatic cells.

Bilirubin, the chief pigment of human bile, is derived, by a process of hydrolysis, from hemoglobin liberated as the result of erythrocyte destruction in the reticuloendothelial system. This pigment is carried in the protein-bound or indirect-acting form (bilirubin-globin) to the liver, where, after the protein has been removed, it is excreted in the bile in the direct-acting or protein-free form, (cholebilirubin). Upon entering the intestine, through a series of reduction processes, due to the action of putrefactive bacteria, it is converted into urobilinogen. A major portion of the urobilinogen is excreted in the feces, a fraction of it enters the enterohepatic circulation and is carried back to the liver where it enters into the formation of more bilirubin, and a third portion enters the general circulation and is eliminated in the urine.

JAUNDICE

Jaundice is a syndrome characterized by hyperbilirubinemia. The concentration of bilirubin in the blood is dependent upon (1) the rate of hemoglobin liberation as the result of erythrocyte destruction, (2) the functional activity of the reticuloendothelial system where bilirubin is formed as a by-product of hemoglobin breakdown, (3) integrity of the hepatic cell whose duty it is to remove bilirubin from the blood, and (4) patency of the biliary canaliculi and bile ducts permitting the normal flow of bile after it has been excreted by the liver.

Thus, Ducci's[3] classification of jaundice would appear to be not only the simplest, but offers the greatest clinical applicability.

1. Prehepatic
   a. Hemolytic
   b. Nonhemolytic
2. Hepatic
   a. Hepatocellular
   b. Hepatocanalicular
3. Posthepatic
   a. Complete obstruction
   b. Incomplete obstruction

Prehepatic jaundice is accompanied by no demonstrable lesion in the hepatic parenchyma and is characterized by a hyperbilirubinemia which gives an indirect van den Bergh reaction since it is bilirubin-globin and has not yet passed through the liver. It may be of the hemolytic or nonhemolytic variety. In the hemolytic type, there is production of bilirubin, due to excessive erythrocyte breakdown, at a rate greater than the hepatic cells can metabolize all of it and, of course, the urobilinogen content of the urine and feces is increased. If the hemolytic process is particularly rapid and severe, hemoglobin and methemalbumin will appear in the plasma in addition to excessive amounts of bilirubin. These three pigments are found in the plasma in all the well-recognized hemoglobinurias such as blackwater fever and nocturnal hemoglobinuria. Hemolytic jaundice may be of the familial or acquired type. In the nonhemolytic type of prehepatic jaundice the rate of bilirubin production is normal but the threshold for concentration in the liver is elevated. This is the "familial nonhemolytic icterus" of Dameshek and Singer.[4] They may be differentiated as follows:

Hemolytic Prehepatic Jaundice	*Nonhemolytic Prehepatic Jaundice*
1. Splenomegaly	1. Normal spleen
2. Spherocytosis	2. No spherocytes
3. Abnormal thickness of rouleaux	3. Normal thickness of rouleaux
4. Increased fragility to NaCl	4. Normal fragility to NaCl
5. Increased fragility to lysolecithin	5. Normal fragility to lysolecithin
6. Reticulocytosis	6. Normal reticulocyte count
7. Increased urobilinogen in urine	7. Normal urobilinogen in urine
8. Greatly increased urobilinogen in feces	8. Normal or low content of urobilinogen in feces
9. Normal result of bilirubin function test	9. Abnormal or delayed result in bilirubin function test.

It is not the purpose of this presentation to differentiate between the numerous causes of prehepatic jaundice in addition to the two familial types already described. Suffice it to say that while they may present many characteristics in common, their differentiation depends greatly upon history, physical examination, and morphological examination of not only the peripheral blood but of the bone marrow as well.

Of the hepatic forms of jaundice, two main types must be differentiated, namely, hepatocellular and hepatocanalicular. The former must be reserved for those cases where there is definite evidence of disturbed function of the hepatic cell. In jaundice of the hepatocellular type there will be hyperbilirubinemia of the prompt direct reacting type and low or absent values for fecal urobilinogen, but abnormal amounts of urine urobilinogen due to the fact that the diseased liver is unable to metabolize the urobilinogen of the enterohepatic circulation. Of course, this elevation of urine urobilinogen is not present if disease is so pronounced that little or no bile reaches the intestinal tract. Tests of liver function which reveal hepatocellular damage will be positive while those of posthepatic jaundice will be negative. If prehepatic hemolytic icterus is present over a period of time, the hepatic damage resulting from the excessive amount of bilirubin and anoxemia will result in hepatocellular damage as revealed by certain tests of liver function. Should the hemolytic disease be of extreme severity

or allowed to persist for a sufficiently long period, hepatocellular damage would be sufficiently extensive to reduce the amount of fecal urobilinogen, thus causing much confusion in differential diagnosis. Herein, although the possibility is remote, hematological findings, pertaining to morphology particularly, would be of diagnostic aid.

The differentiation of hepatocellular and hepatocanalicular types of jaundice is of utmost importance. It has been repeatedly shown that many cases of jaundice originating in the liver itself give no demonstrable evidence of hepatic cell involvement. To go a point further, many of these cases actually present evidence of obstructive jaundice. Indeed, these characteristics have not only been noted in many cases of jaundice following administration of arsenicals, sulfonamides, and thiouracil but in cases of hepatitis as well.[5] Histologic changes, such as periportal cellular accumulations, bile thrombi, or actual increase in size of the liver cords, often appear inadequate to account for the jaundice on the basis of cholangiolitic obstruction. It is believed that the continued regurgitation of bile supports the concept of increased permeability (functional injury) of the canaliculi.

In certain cases of hepatitis, liver biopsy may fail to reveal histologic evidence of appreciable hepatocellular injury at a relatively early stage. Since in these cases there is often some residual evidence of reduced hepatocellular function, it is believed that there was, initially, a more marked liver cell injury, and that prolonged regurgitation jaundice simply indicates a more severe affection of the intrahepatic bile duct system than in the ordinary type. The transition of this form of hepatitis to cirrhosis suggests that the term cholangiolitic cirrhosis is more appropriate and descriptive than hypertrophic biliary cirrhosis. The end stages of the cholangiolitic cirrhosis following prolonged hepatitis may be indistinguishable from atrophic or portal cirrhosis. In cases of hepatocanalicular type, the hyperbilirubinemia will be of the direct or prompt reacting type, there will be a decreased flow of bile in biliary drainage, the stools will be of the acholic type containing no urobilinogen, while the urinary values for urobilinogen will be normal, and the urine will contain bile pigments and salts. Tests of hepatocellular function will be normal, while those tests for posthepatic obstruction will be positive.

A few cases of jaundice will give clinical and laboratory evidence of hepatocellular and hepatocanalicular involvement at the same time. These cases represent merely a transition of one form to the other and the diagnosis may only be established with the aid of biopsy or peritoneoscopy.

Posthepatic jaundice of the incomplete type will show the hyperbilirubinemia to be of the direct acting type and because of the valvular nature of the obstruction, these values will fluctuate. Urobilinogen content of the feces will show a corresponding fluctuation and even though a very small amount of bilirubin is permitted to reach the duodenum, the urobilinogen thus formed will not be taken up by the secondarily involved liver and abnormal values for urine urobilinogen will be noted with the additional presence of bile pigments and bile salts. Should biliary drainage be used as a differentiating test, it may prove quite confusing because instead of obtaining an acholic specimen, it may well be laden with large amounts of hyperconcentrate A bile due to its release by the passage of the tube.

In complete obstructive posthepatic jaundice, as seen in carcinoma of the head of the pancreas and ampulla of Vater, the hyperbilirubinemia is of the direct reacting type and of very high concentration. There is a persistent nonfluctuating type of acholic stool with almost complete absence of urobilinogen. The urine will contain negligible amounts of urobilinogen but will be heavily laden with large amounts of bile pigments and bile salts. Theoretically, at least, in both types, there will be negative tests for hepatocellular damage and positive results for tests of posthepatic obstruction.

The primary value of any classification is to present a logical schema to aid in understanding fundamental mechanisms and pathogenesis. So it is with the classification of jaundice, for, as it must be apparent from the foregoing discussion, rarely do we see cases early enough to obtain clear-cut studies of liver function. In many instances, prehepatic jaundice presents no particular difficulty in differential diagnosis. There are those cases, however, representing a transitional stage which will tax the ingenuity of the most astute clinician. Differentiation of hepatic and posthepatic jaundice is usually more difficult and, of course, extremely important because in the one instance the therapeutic approach is medical and in the other surgical. An expert history and physical examination, plus serial studies of liver function, and the possible aid of liver biopsy offer the only possible chance of correct diagnosis in the majority of cases.

TESTS OF LIVER FUNCTION

In a recent article by Bruger and Oppenheim[6] some 55 various tests of liver function have been enumerated. To endeavor to employ such a battery of tests would not only be cumbersome, impractical, and unnecessary but also illogical. Therefore, it is the purpose of this presentation to review the simpler and more useful tests pertaining to liver function and finally to offer a logical approach in their application in the differential diagnosis of hemolytic disease and hepatic insufficiency.

Bilirubin in the Serum.—The quantitative estimation of serum bilirubin is the most accurate index to the degree of jaundice. It obviously offers no aid in differential diagnosis, but is an aid in following the course of prehepatic, hepatic, and posthepatic mechanisms. Elevation above 1.2 mg. per cent signifies retention of bilirubin in the blood or its regurgitation from the small biliary passages into the blood. In prehepatic jaundice, the degree of bilirubinemia aids in determining the degree of hemolysis for, after a hemolytic crisis, it will reach its peak in from 3 to 5 hours and its decline will precede the reticulocytosis when the hemolytic process is going into remission. While hyperbilirubinemia is not a distinguishing characteristic of liver disease per se, the determination of serum bilirubin may aid in following its course. In jaundice of posthepatic origin, the level of serum bilirubin is not of as much importance as the constancy of elevation. Fluctuation suggests incomplete obstruction, while a persistent or increasing concentration suggests complete obstruction.

Icterus Index.—The icterus index is a nonspecific guide to the degree of jaundice inasmuch as it is only a comparison of the yellow color of the serum to an arbitrary standard which is usually a 1:50,000 solution of postassium dichromate. Any substance that will impart a yellow color to the serum, such as

carotene, nicotinic acid, riboflavin, and traces of hemoglobin will elevate the icterus index. As a rough clinical guide, it has its place, but when in doubt as to the presence or absence of jaundice, serum bilirubin estimation is the only reliable test.

Van den Bergh Reaction.—The van den Bergh reaction determines only the form in which bilirubin exists in the blood stream. Bilirubinglobin derived from the breakdown of hemoglobin in the reticuloendothelial system gives the indirect reaction while bilirubin derived from the breakdown of bilirubinglobin in the hepatic parenchymal cell gives the direct reaction. It follows, therefore, that a direct van den Bergh reaction is present in both hepatic and posthepatic jaundice and that its determination is of no aid in differentiating hepatocellular and obstructive mechanisms whether intrahepatic or extrahepatic. In prehepatic icterus, it has its primary value because only in this condition is the increased amount of bilirubin in the blood of the indirect acting type with normal values for the direct acting bilirubin.

Bilirubin in the Urine.—During an epidemic of infectious hepatitis, Gellis and Stokes[7] developed a modified methylene blue test for the detection of bilirubin in the urine. It was found useful in detecting the presence of this pigment in the preicteric state of infectious hepatitis. It may also be utilized as a screening procedure for the detection of early hepatic involvement in individuals exposed to certain industrial poisons. Its reliability has been questioned since the test is not specific for bilirubin. Yellow urine from normal persons and yellow substances such as potassium dichromate or ferric chloride will produce a green color when added to methylene blue. The Harrison spot test and the barium strip modification may also be used to detect bilirubinemia.

Urinary and Fecal Urobilinogen.—It must be remembered that for urobilinogen to be formed, bilirubin must enter the intestinal tract. It follows, therefore, that in prehepatic hemolytic icterus, the quantity of urinary and fecal urobilinogen will be increased. Their 24 hour quantitative estimation is of great value. In familial nonhemolytic prehepatic jaundice, the urobilinogen content of the urine and feces will be within normal limits. In hepatic jaundice of the hepatocellular type, there will an absence of urobilinogen in the feces but the urinary content will reach pathological levels because the liver will be unable to metabolize the urobilinogen carried to it in the enterohepatic circulation and it will, therefore, be eliminated in excessive amounts. If the disease is so extensive that no bile enters the duodenum, urinary urobilinogen will be decreased or absent.

If the integrity of the hepatic cell is maintained and the jaundice is of the hepatocanalicular type, fecal urobilinogen will be absent, and even though very little bile is reaching the duodenum, normal values for urinary urobilinogen will prevail. In incomplete obstructive posthepatic jaundice, fecal urobilinogen fluctuates from very low to abnormal values corresponding to the degree of hyperbilirubinemia. The urinary urobilinogen is usually abnormal and fluctuating if any bile is reaching the duodenum; this results from the hepatocellular damage which is usually present in individuals with long-standing biliary disease. In complete obstructive posthepatic jaundice there is absence of fecal urobilinogen and very low values for urinary urobilinogen. According to Bruger and Oppenheim[6] the modified 2 hour test for fecal and urinary urobilinogen as described by Watson has proved to be a satisfactory quantitative procedure.

Bilirubin Excretion Test.—It is well recognized that there are many cases of liver damage without hyperbilirubinemia. It has been found that when bilirubin is injected intravenously into normal individuals in a dosage of 1 mg. per kilogram of body weight, it is almost completely eliminated from the blood stream in 2 to 4 hours. If more than 6 per cent of the injected dye is retained in the blood at the end of 4 hours, the ability of the liver to excrete the pigment is impaired. As pointed out above, this test is not only of value in determining slight hepatic damage in the absence of jaundice but is of value in differentiating prehepatic hemolytic icterus from prehepatic nonhemolytic icterus, normal values often being obtained in the hemolytic type and retention in the nonhemolytic type.

Bromsulfalein Excretion Test.—Five mg. of the dye per kilogram of body weight is injected intravenously. The dye is removed from the blood by the Kupffer cells and excreted by the parenchymal cells into the bile. A normal liver will allow no more than 4 per cent retention of the dye in 45 minutes if a photoelectric colorimeter is used. It is a very sensitive, reliable, safe test but of very limited value in the presence of jaundice.

Oral Galactose Tolerance Test.—Galactose is the sugar selected to estimate the carbohydrate function of the liver because it is rapidly absorbed, does not require insulin for its metabolism, and has no renal threshold. Forty gm. of galactose is dissolved in 400 cc. of water and taken by the patient in a fasting state. The urine is collected for 5 hours and a quantitative estimation of urine sugar is done. Normally no more than 3 gm. of sugar are excreted in this interval. The presence of impaired renal function introduces a considerable error. This test has the added disadvantage of being very expensive. It is positive in acute and chronic hepatic parenchymal disease and may be used in the presence of jaundice to differentiate hepatocellular, hepatocanalicular, and posthepatic obstructive jaundice.

Total Cholesterol and Cholesterol Partition.—When employing the Bloor technic, normal values for total serum cholesterol range from 160 to 230 mg. per cent. The cholesterol esters range from 60 to 120 mg. per cent. The ratio of combined to the total cholesterol is relatively constant ranging between 40 and 60 per cent. Since the liver has for one of its functions the esterification of cholesterol, in hepatocellular disease the ester fraction is markedly reduced or entirely absent. Hanger[8] feels that in individuals exposed to industrial hepatotoxic agents serial observation of the cholesterol esters offers the most reliable index to the early development of hepatocellular disease. The total cholesterol values remain unchanged. In obstructive jaundice of either the hepatocanalicular or posthepatic type, the total serum cholesterol values are elevated. This is not due to the damming of the cholesterol by obstruction but to the absence of bile salts which are necessary for the excretion of cholesterol via the intestinal mucosa, thereby disturbing the normal equilibrium between synthesis and excretion of this substance.

Morphology of the Erythrocytes.—In the jaundiced patient, a macrocytic, hyperchromic anemia is characteristic of extensive parenchymal cell damage

which results in the failure to store the erythrocyte maturing factor. Spherocytosis is characteristic of familial hemolytic anemia. All other hemolytic anemias are normocytic and normochromic with the exception of Mediterranean anemia which is microcytic and hypochromic and in which target cells are present. Marked poikilocytosis and anisocytosis are of course present in sickle cell anemia. Occasionally in extensive hepatic disease a clumping of the red cells in Hayem's solution will be noted. This is probably due to precipitation of the elevated globulins by the mercuric chloride in the diluting solution. In addition to advanced cirrhosis, this phenomenon is noted in kala-azar, nephrosis, multiple myeloma, et cetera.

Serum Albumin.—Tumen and Bockus[9] found that a reduction in albumin was the most consistent alteration in the serum proteins in patients with chronic advanced liver disease. Lowering of the albumin-globulin ratio was not found to be as constant or as significant as a reduction in the serum albumin. The rise of serum albumin during clinical improvement in patients with cirrhosis is a frequent finding. Persistent or decreasing serum albumin concentration is a poor prognostic sign.

Cephalin-Cholesterol Flocculation Test.—The serum of patients with parenchymal cell damage has the ability to flocculate a cephalin-cholesterol emulsion. It is believed that gamma globulin is the substance which causes this flocculation and under normal circumstances the process is inhibited by the presence of serum albumin. The alteration in albumin, the result of liver cell damage, reduces its capacity to prevent this flocculating action on the part of the gamma globulin. Readings of 2 plus to 4 plus are considered abnormal. This test is by no means specific for hepatic disease since it is positive in a variety of other clinical disorders. It is a very delicate test and unless carefully controlled is subject to great error.

Thymol Turbidity and Flocculation.—As with the cephalin-cholesterol flocculation test, the thymol turbidity test does not test any known function of the liver and should be regarded as an indicator of disturbed liver metabolism rather than a function test. Sera from patients with active parenchymal cell damage, when added to a saturated solution of thymol buffered with barbital and sodium barbital at a pH of 7.8, produce turbidity. However, the mechanism is somewhat different than that of the cephalin flocculation since it is dependent upon certain lipids and some abnormal lipoprotein associated with the beta globulin fraction of the serum.[8] Various methods for quantitative evaluation of the degree of thymol turbidity have been suggested but the original method proposed by Maclagan[10] utilizing the standards devised by Kingsbury and his associates[11] provides a clinically accurate method. When Maclagan originally described the test, he noted that if certain solutions giving positive results were allowed to stand overnight, flocculation occurred. He attached no particular significance to this, but Neefe[12] noted that the thymol flocculation remained positive for some time after the thymol turbidity returned to normal values. In addition to supplementing the thymol turbidity test, the presence or absence of 2 plus to 4 plus flocculation at the end of 18 hours may aid in evaluating the significance of borderline or weakly positive thymol turbidity readings. Positive findings are particularly prominent in infectious hepatitis and cirrhosis.

In acute hepatitis, the cephalin-cholesterol flocculation test usually becomes positive earlier than the thymol turbidity but the latter remains positive longer during convalescence. The cephalin flocculation may give a false positive result in aged serum, whereas the thymol turbidity is negative. In neoplasms of the liver, the cephalin flocculation is usually negative and the thymol turbidity may be positive. The cephalin flocculation is not infrequently negative with lipemic serum and in this instance, the thymol turbidity will be positive.[8] From the foregoing, it is obvious that neither the thymol turbidity nor the cephalin flocculation supplant each other but rather supplement each other and should be performed in combination.

Colloidal Red Test.—This test[13] is performed in the same manner as the colloidal gold test except that, instead of colloidal gold, a colloidal suspension of scarlet red is used. It is sensitive, depending probably on some alteration in the gamma globulin, and closely parallels the cephalin flocculation and thymol turbidity tests.

Zinc Sulfate Precipitation.—Kunkel,[14] cited by Bruger and Oppenheim, reported that when serum with abnormally high gamma globulin was diluted with a solution containing a small amount of zinc sulfate, a turbid precipitation occurred, the optical density of which was proportional to the degree of hyperglobinemia. Normal serum does not flocculate for at least 12 hours whereas serum with abnormally high concentration of gamma globulin usually flocculates within 4 hours. While it is far from a specific test, it is found useful in determining the alterations in gamma globulins during the course of infectious hepatitis and of particular value in detecting chronic liver disease following infectious hepatitis.

Prothrombin Determination and Response to Vitamin K.—The formation of prothrombin by the liver is dependent upon the ingested vitamin K. Bile salts are necessary for the absorption of vitamin K from the small bowel. In the writer's hands, the Link-Shapiro modification of the Quick method of prothrombin determination has been the most satisfactory. Utilizing this method, normal values vary between 14 and 16 seconds for undiluted plasma. In the presence of extensive hepatocellular disease, if the prothrombin time is elevated, it is caused by the inability of the liver to form prothrombin, and parenterally administered vitamin K will produce no change in these values. However, elevated prothrombin time, in the presence of hepatocanalicular or posthepatic jaundice, results from the absence of bile salts in the small bowel preventing vitamin K absorption; in the vast majority of cases the prothrombin time will revert to normal following parenteral administration of the vitamin.

Hippuric Acid Synthesis.—Hippuric acid is synthesized in the liver by the union of the benzoate radical with glycine. When 6 gm. of sodium benzoate are given orally, the normal liver can produce 2.5 to 3 gm. of hippuric acid within 4 hours. The excretion of less than 2.5 gm. of hippuric acid in the urine in 4 hours signifies hepatic damage providing renal function is not impaired. The intravenous administration of hippuric acid merely renders the test more sensitive. Hippuric acid synthesis is reduced in acute and chronic parenchymal disease and in metastatic carcinoma. It is normal in obstructive jaundice providing cirrhosis is not present from long-standing obstruction.

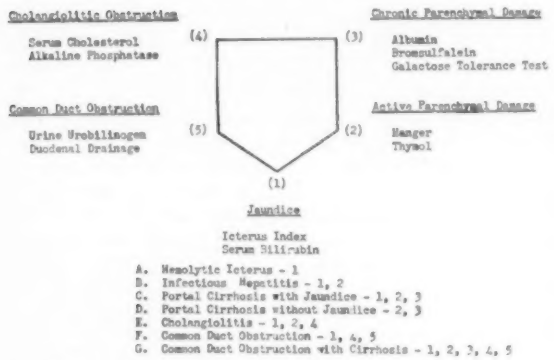

Fig. 1. Schema for coordination of liver function tests, after Bruger.[13]

Serum Alkaline Phosphatase.—Whether or not alkaline phosphatase originates in the liver has not been clearly established; however, it is contained in the bile. In hepatocanalicular or posthepatic obstructive jaundice, serum alkaline phosphatase is elevated. It may also be elevated in hyperparathyroidism, Paget's disease of bone, and metastatic carcinoma of bone. However, values above 13 units in an adult with jaundice are indicative of an obstructive lesion.

Sedimentation Rate.—As correlated with liver biopsies, sedimentation rates above 15 mm. per hour in the presence of liver disease, were concluded by Popper and others[15] to be indicative of inflammatory change in the portal triads, provided other inflammatory foci were eliminated.

Serum Nonprotein Nitrogen.—Values above 40 mg. per cent may be associated with liver damage and are due to increased reabsorption of nitrogenous substances in the renal tubules. Primary renal disease must be excluded. Such pathological reabsorption occurs more often in hepatocellular disease than in any other type.[15]

SUMMARY

Greene's schema for the co-ordination of liver function tests as modified by Bruger[13] (Fig. 1) affords an excellent system to aid in their intelligent and logical application.

Liver and biliary disease may be analyzed by the presence or absence of five factors; namely, jaundice, active parenchymal damage, chronic parenchymal damage, hepatocanalicular obstruction, and posthepatic obstruction.

The presence or absence of jaundice is confirmed by serum bilirubin estimation and the icterus index. In active parenchymal damage, the cephalin-cholesterol flocculation and thymol turbidity tests will be positive. The presence of chronic parenchymal damage will be denoted by bromsulfalein retention in the absence of jaundice, and in the presence of jaundice by an abnormal galactose tolerance test. In hepatocanalicular jaundice, total serum cholesterol and serum alkaline phosphatase values will be elevated. In posthepatic jaundice, there will be an absence of urobilinogen in the urine and bile in the biliary drainage.

In considering the applicability of these tests to various hepatic disorders it is seen at once that in hemolytic disease, there will be an elevated serum bilirubin (indirect reacting) and icterus index, but the other four factors will be undisturbed. The cephalin flocculation and thymol turbidity will be negative. In cirrhosis of the liver with jaundice, factors 1 and 2 will be abnormal and the galactose tolerance test will be positive. In cirrhosis without jaundice, factors 1 and 2 will be positive with retention of bromsulfalein. With or without jaundice, values for serum albumin will be decreased. In hepatocanalicular jaundice, the serum bilirubin and icterus index is elevated, there may or may not be evidence of active hepatocellular damage, but the total serum cholesterol and alkaline phosphatase will be increased above normal values. In common duct obstruction, factors 1 and 4 will be positive with absence of urine urobilinogen and bile in biliary drainage. In hepatocanalicular or common duct obstruction with cirrhosis, all five factors will be disturbed as evidenced by all positive tests. In carcinoma of the liver, whether primary or secondary, the pattern will obviously be quite irregular.

CONCLUSIONS

1. The mechanism and classification of jaundice have been presented.

2. It has been pointed out that rarely do the diseases, which have jaundice in common, exist as distinct entities.

3. Various liver function tests have been presented and their relative merits discussed.

4. A simple plan for coordinating liver function tests has been presented.

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Treatment of Hypertension

Part I. The Osteopathic Internist's Viewpoint on Present-Day Management of Hypertensive Vascular Disease*

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Interest in the treatment of hypertensive vascular disease is not new to the healing arts professions, nor is it limited to them. The frequency of occurrence and the consciousness of the public of hypertension are two of the factors which keep the disease foremost in mind. But, in spite of widespread interest, we are still at sea as to the cause of many cases of hypertensive vascular disease and, therefore, we lag in treatment as well. As a result many forms of therapy have been and are being used, but no one of them has proved unquestionably superior to any other. Only a few have been shown to be effective and even these are not consistent in their effects.

There are certain factors, such as kidney and endocrine malfunction, that will produce an increase in the blood pressure. When these factors are eliminated early in the course of the disease the pressure will usually return to normal. However, by far the largest number of cases of hypertensive vascular disease are a result of unknown cause or causes. It is primarily this group that is difficult to treat adequately and most of the presently used forms of therapy are directed toward the correction of this type of hypertensive disease. The discussion in this paper will deal only with therapy as related to hypertensive vascular disease of unknown etiology.

The methods of treatment that have been used are too numerous to mention; however, they may be classed under five major headings: (1) Osteopathic manipulation, (2) drugs, (3) diet, (4) roentgen irradiation, and (5) surgical intervention. Clinical reports have been published regarding the various types of treatment but a true evaluation of any one or all is difficult.

Blood pressure is influenced by various factors, among which the psychogenic is one of the most important. The level of pressure may show wide variations and the psychogenic factor plays a large part in producing the changes. This point can be emphasized by a personal experience. A male patient had been under treatment with a significant lowering of pressure and relief of symptoms. However, upon learning that the patient became apprehensive before each visit about the pressure status that would be found that day, a system was evolved to minimize the effects of his apprehension. Immediately upon the patient's entrance into the office the blood pressure was checked. After spending the next 3 or 4 minutes talking about whatever came to mind, I checked the pressure again and found that there was always a drop of at least 10 mm. and on one occasion there was a drop of 20 mm. From this example it can be seen that even if a number of readings were used the average would still not be a true basal level.

If the pressure of an individual who is clinically responding to treatment can vary to such an extent as that of my patient, then certainly the pressure of one who is being evaluated for treatment can do the same. In fact the pressure of these patients may vary more because they are under the added emotional and mental strain of starting a new course of therapy whose outcome they may question. It does not matter whether or not the patient is hospitalized since the same factors are present in every case, although they may vary as to degree. One cannot stop the patient's mental and emotional processes nor prevent their influence on the circulatory picture. Therefore, it cannot be assumed that the basal levels found in the statistical surveys are a true indication of the degree of hypertension. The blood pressure levels observed following therapy are subject to the same criticism. Some individuals will respond favorably regardless of the type of therapy because they are psychologically receptive to being helped or possibly their faith in the physician is enough to relieve their emotional fears.

One other factor to be taken into consideration in evaluating forms of therapy is the criterion of improvement. Little attempt has been made to standardize results, making comparison extremely difficult, but regardless of the possibility of error, any attempt at evaluation of present-day forms of therapy must use available statistics as a guide.

OSTEOPATHIC MANIPULATION

Every general practitioner knows the value of osteopathic manipulative therapy for hypertensive vascular disease. Eggleston's[1] findings chart the effects of such therapy on the systolic, diastolic, and pulse pressures in lesioned and nonlesioned patients. Detwiler[2] confirmed Eggleston's findings in a report covering a significant number of patients. Long and Deming[3] found that mobilization of the upper dorsal area would lower blood pressure. The mechanism by which the blood pressure is lowered is still not definitely known; however, the action is probably by way of the autonomic nervous system. There is no question of the fact that some of the response may be psychological.

Regardless of the mode of action, it has been my experience that most hypertensive individuals will respond, at least to some extent, to osteopathic manipulation. The response in many patients is immediate. How long this effect lasts is not known but with continued treatment the decrease in blood pressure tends to become constant in a large number of cases. I feel that the percentage of response to manipulative therapy is as good as that to any other form of present-day management.

Osteopathic manipulation obviously has much in its favor when we stop to realize that our results are obtained without toxic side effects. Drug therapy as it stands today has definite value but unfortunately those agents which influence the hypertension are apt to be severely toxic. While it is possible for an individual to be hyper-reactive to manipulation, he is never in severe danger. The response of the individual and the lack of toxic effects indicates that osteopathic

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manipulative therapy is definitely one of the better forms of treatment of hypertensive vascular disease today.

DRUG THERAPY

Recently there have been reevaluations of many of the older drugs and the introduction of many new ones for the treatment of hypertensive disease. Because the list of drugs being used is fairly long, this paper will attempt to touch on only the more recent and the most effective.

Potassium thiocyanate, while not a new drug, is used frequently today. The chief objection to its use in the past was the danger of toxic reaction but with present methods of control it has been used with varying degrees of success and it can, with discretion, be administered by the general practitioner. By its use both the systolic and the diastolic pressures may be appreciably lowered with relief of symptoms. However, symptomatic relief has also been reported without any lowering of the blood pressure.

The exact mode of thiocyanate action has been controversial but it has been found recently that the hypotensive effect is a result of renal tubular regulation of electrolyte excretion and that its action is enhanced by the simultaneous use of the mercurial diuretics.[4] The toxicity of the drug necessitates rigid control of the blood level to prevent the appearance of toxic symptoms. Administration of potassium thiocyanate in the presence of increased capillary fragility is apt to be dangerous and various types of vascular accidents have been reported following its use in patients with that condition.[5] Regardless of the possible complications, the use of potassium thiocyanate is warranted in the treatment of some cases of hypertension and with careful control of dosage, adverse reactions may be avoided.

Veratrum viride is another older drug that has recently found favor in the treatment of hypertension. Again we are dealing with a toxic drug which makes administration difficult. However, it has been found that the hypotensive action may be obtained before toxic levels are reached. Various actions of the drug are known but how the hypotensive action is produced has not been ascertained. Published results of the use of this drug are variable.[6] Approximately 10 to 20 per cent of patients fail to respond at all,[7] and in those who do respond the reaction is extremely variable. Usually the response is more effective when the drug is used only for short periods. At present there is no laboratory method of maintaining accurate dosage; therefore, each individual must be closely observed for the appearance of toxic symptoms. Although an extremely toxic drug, veratrum viride, like potassium thiocyanate, may be used with satisfactory results.

Rutin, a nontoxic substance, several years ago received favorable notice for its hypotensive qualities. It is now known that although there is a hypotensive action its main advantage lies in its ability to combat the increased capillary fragility which so frequently accompanies hypertensive vascular disease. Griffith and Lindauer[8] have shown that increased capillary fragility can be returned to normal in a large percentage of cases with the administration of rutin. Since increased capillary fragility makes an individual more prone to vascular accidents, rutin is being used routinely in many cases of hypertension. At the present time this appears to be good therapy. Rutin should definitely be used during therapy with potassium thiocyanate.

Various other drugs, particularly sympathicolytic agents, are being used in present-day therapy. Tetraethylammonium salts are probably the best known example. Lowering of blood pressure has been demonstrated with the use of these salts but the action is temporary.[9] The use of these agents is not without danger of toxic reaction; therefore, their use at the present should be limited to the more serious emergencies. Other sympatholytic agents are still in the experimental stages and will not be discussed.

DIET

An increase in interest in the effect of diet on hypertension has been evident in the past few years. Kempner's rice diet and the sodium free diet are discussed in Part II beginning on the next page.

ROENTGEN THERAPY

X-ray therapy has been used in the treatment of certain cases of hypertension, with impressive results. The exact mechanism for the action of roentgen therapy is not known but it is possible that the response is obtained in much the same manner as with sympathectomy. However, x-ray therapy has the added advantages of repeated courses of therapy and the patient does not have to be subjected to the fear of surgery.

Paul T. Lloyd[10] has treated a series of cases by this method. Satisfactory results were obtained in approximately 85 per cent of cases. Hypertension accompanied by arteriosclerosis or renal deficiency will not respond to irradiation. The best results have been obtained in individuals who were in the menopause.

SURGICAL THERAPY

Sympathectomy in the past was reserved for hypertensive vascular disease that was far advanced. The tendency now is to use surgery much earlier in the course of the disease, before the process becomes irreversible. Many authorities feel that the poor results obtained previously might have been better if the treatment had been instituted earlier. Evelyn, Alexander and Cooper[11] recently reported on a follow-up of 219 cases. Five years after operation there was a reduction in the blood pressure to normal in 8 per cent and a significant reduction, but not to normal, in 13 per cent. Of the remainder 52 per cent showed no significant reduction and 27 per cent were dead. Although there is no significant response of blood pressure in the largest number of surgically treated cases, there is definite symptomatic relief. As a result most of the patients are rehabilitated for a time. The period of effectiveness at present is relatively short. Perhaps with the evolvement of new technics and with earlier intervention, the response of hypertensives will be more favorable.

SUMMARY

Review of present-day management of hypertensive vascular disease reveals that as yet the fundamental answer to the treatment of this condition has not been found. Clinical results indicate that we, as osteopathic physicians, have one of the more effective forms of therapy at our command. Manipulative therapy should be instituted in all cases of hypertension and other forms of therapy instituted as indicated. Until more results are available, surgical intervention should be reserved for those individuals who did not respond to the more conservative types of therapy.

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Part II. Evaluation of the Use of Salt Free Diet and Rice Diet in Arterial Hypertension*

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The treatment of hypertensive vascular disease by lowering the sodium chloride intake was recommended as early as 1904 by Ambard and Beaujard.[1] However, this form of therapy did not come into general use. Kempner's[2] use of the rice diet which is low in salt content as well as protein and fat has caused a renewed interest in the treatment of hypertension by the use of dietary restrictions. Excellent results with the use of a salt free diet alone have been reported. The results obtained with either the rice diet or the salt free diet have been extremely variable and because of this, evaluation of clinical results is difficult.

The factor that probably is most responsible for making the evaluation of the results of dietary management of hypertension so difficult is the lack of uniformity in the recording of blood pressure. Pressure readings vary with the individual who is eliciting the blood pressure and of course the pressure varies with the patient's position, time of day, activity, et cetera. These factors have not been standardized by some research groups and therefore the results obtained may be questioned. Also different researchers have used various methods of arriving at a basic blood pressure level. Because of this fact, it is almost impossible to compare results and arrive at definite conclusions as to the value of either the rice diet or the salt free diet.

Another factor entering into the evaluation is the standard of improvement. Some workers use a drop to 150 systolic and 100 diastolic or below as a sign of definite improvement. Others use significant drops in systolic and diastolic pressures as their criteria. Symptomatic improvement of the patient regardless of the fall of the blood pressure also is considered. Until the criteria necessary for improvement are standardized, a true evaluation of results will be impossible.

RICE DIET

Kempner[2] reported successful results with the rice diet in the treatment of hypertensive heart disease in 1944. The diet as used contains 2000 calories with no more than 5 grams of fat and 20 grams of protein. The patient is given an average of 250 to 350 grams, dry weight, of rice daily. The rice is prepared by boiling and without the addition of salt. Practically all fruits and fruit juices are allowed, but nuts, dates, and avocados are excluded. The total fluid intake for 24 hours is limited to 700 to 1000 cc. Patients remain on this diet for an average of 3 months, unless they show a satisfactory drop in pressure before that time. At the end of the 3 months the diet is gradually modified to include other foods.

In 1948 Kempner[3] published another report on the results obtained in treating hypertensive vascular disease by means of the rice diet. This report states that of the 500 patients included in the report, 322 or 64.4 per cent showed improvement. Improvement in these cases was based on the following factors: drop of at least 20 mm. in the mean arterial tension, reduction in the heart size, disappearance of retinal pathology, and changes in T1 lead of the electrocardiogram.

Kempner also pointed out that while the blood urea nitrogen and nonprotein nitrogen values are higher than normal when fasting they are lower than normal when the rice diet is utilized. The hemoglobin and plasma protein levels are maintained on the rice diet. The sodium content of the urine decreased 99 per cent while the sodium content of the blood decreased 4.3 per cent. The chloride concentration of the urine decreased 96 per cent while the chloride concentration of the blood decreased 5.6 per cent. The potassium content of the urine increased 78 per cent and the potassium content of the blood increased 0.8 per cent.

Loofbourow, Galbraith, and Palmer used the diet in a series of 56 patients.[4] Of the 56 only 16 stayed strictly on the diet and 20 followed it moderately well. Using the Kempner method of mean blood pressure (systolic plus diastolic divided by 2), 45 per cent of the patients showed improvement. Using a drop in blood pressure to 150/100, 25 per cent of those who adhered strictly demonstrated improvement and 20 per cent of those who adhered moderately showed a favorable response.

In 1948, Contratto and Rogers[5] reported a series of cases, using the rice diet as a form of therapy. They began with 55 patients but of this number 18 were excluded because they did not remain on the diet. Of those that maintained their diet, 43 per cent showed a drop in pressure to 150/100 or below. Eight other patients had a systolic drop of 50 or more and a diastolic of 20 or more. The urinary chlorides in this series of cases averaged 8.6 gm. before beginning the diet and dropped to 1 gm. in the first 2 weeks and to less than 0.5 gm. in 6 weeks. None of the

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patients in this group developed a sodium or chloride blood level deficiency.

In my own experience, I have had only one patient who has followed the rice diet faithfully. The drop in blood pressure, however, has been only 50 mm. systolic and 15 mm. diastolic. The diet of this patient has been modified, of late, without any noticeable change in the blood pressure reading.

The mechanism by which the blood pressure is lowered by this diet has not been definitely established. Grollman and his associates[6] believe that the drop in pressure is a result of the low sodium chloride content of the diet. They also point out that patients on the rice diet must be closely observed for signs of sodium chloride deficiency. Mosenthal[7] points out that any procedure which causes a lowering of the nitrogen content of the body will result in the lowering of the blood pressure. That the Kempner diet is low in nitrogen content, 2.63 gm. per day, was revealed in studies done by Schwartz and Merlis.[8] They found that after 8 days of the rice diet all patients had a negative nitrogen balance. There was an average deficit of 3.23 gm. of nitrogen per day. If this be the mechanism of the lowering of the blood pressure, it is a more or less dangerous one.

The value of using the rice diet in the treatment of hypertensive heart disease, in my opinion, is questionable. The first point of contention is whether the cure is not worse than the disease. It has been my experience that the majority of individuals would rather have the high blood pressure than undergo the strenuous dietary therapy. Another objection of many of the patients is to the frequent check ups and laboratory examinations that are necessary. This factor alone may have a definitely poor psychological effect upon the patient. While the low sodium content and the negative nitrogen balance may be responsible, alone or together, for the lowering of the blood pressure, it is definitely dangerous and contrary to all the teachings of normal physiology to maintain these states for a long period.

It has been observed that drastic food restriction will usually result in the lowering of the blood pressure in both normal and hypertensive individuals. At the same time, the cardiovascular dynamics are so disturbed that any sudden change of the diet may result in congestive heart failure.[9] This fact should be kept in mind when using the rice diet because there are many individuals who will stay on the diet for a number of weeks and then suddenly return to their normal diet.

SODIUM CHLORIDE FREE DIET

While the salt free diet is probably one of the more frequently used forms of therapy in hypertensive vascular disease, it is of doubtful value. As yet there have been no thoroughly substantiated results reported on a long series of cases.

Hypertensive heart disease is accompanied by some disturbance in salt and water metabolism. With this fact in mind, it would seem logical that a low salt diet might be of benefit in the treatment of hypertensive disease. Grollman and Harrison[10] found that a rice diet reduced blood pressure, experimentally in rats, and also observed that this effect could be countered by the addition of sodium chloride to the diet. Perera and Blood[11] found that a restriction of sodium chloride may lower blood pressure. They also observed that large amounts of salt could exert a pressor effect upon individuals who had hypertensive heart disease. Another of their findings was that sodium chloride aids in the activation of desoxycorticosterone acetate. This last may be the mode of action in the lowering of the blood pressure; it certainly may be one of the answers as to why some of the patients respond and others do not.

Sodium restriction for hypertension was used by Dock[12] with about a 50 per cent response. Schroeder[13] reported a drop of 20 mm. or more in only 17 per cent of the cases treated. Bryant and Blecha[14] used a low salt diet on a series of 100 cases and reported improvement in 58 per cent.

While a sodium free diet may be beneficial in the treatment of hypertensive disease, this form of therapy is not without its dangers. The development of uremia as a result of sodium depletion, particularly in the older age groups and in those individuals having concomitant renal disease, is not infrequent.[15] Symptoms due to the lack of salt may occur as well as shock in individuals with severe renal damage. A negative nitrogen balance might occur because of the loss of desire for food.

Again, in the salt free diet as in the rice diet, it is extremely difficult to get the patient to cooperate fully. Most individuals use a considerable amount of salt to season their food and complain bitterly of the taste of food cooked without salt. Another problem is the preparation of salt free diets, particularly for one member of the family. It has been my experience that at least one of these factors is always mentioned in complaints concerning the diet. Many patients will cease their complaints after staying on the diet for several weeks.

As I stated at the beginning of this discussion of the salt free diet, its value in the treatment of hypertension is questionable. This is particularly true since the results of moderate restriction are comparable with those of rigid restriction.[16] There is not sufficient evidence as yet as to the effect of rigid salt restriction upon hypertensive heart disease.[17] I have used moderately restrictive diets in the treatment of hypertensive patients. However, these diets have always been used in conjunction with other forms of therapy, particularly regular osteopathic manipulation. I am, therefore, in no position to say whether the improvements noted were due to the elimination of salt, to one of the other forms of therapy, or to a combination of two or more. Schroeder[13] found that many subjects responded favorably to low salt diets, but he felt that a psychogenic factor played a major role in some instances.

SUMMARY

The efficacy of the rice diet or the salt free diet in the treatment of hypertensive heart disease has been questionable from the time they were first introduced.

The dangers of the rice diet are negative nitrogen balance, sodium deficiency, and possible congestive heart failure. The low sodium chloride diet may result in sodium deficiency, shock, or possibly uremia.

Both diets are difficult for the patient to follow and are apt to depress the patient psychologically. Of the two forms of diet the sodium free is probably the most palatable and the easier to follow.

The use of either the rice diet or the sodium free diet in mild states of hypertensive disease is apparently of little benefit. Certainly a large majority

(Continued on page 291)

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THE MIDYEAR MEETING OF THE BOARD OF TRUSTEES

Meeting at the time of another national emergency, the Board of Trustees of the Association, in session in Chicago December 13, 14, 15, and 16, gave its attention to the effect of the emergency on the manpower of the Nation, most particularly in relation to the utilization of osteopathic physicians and surgeons, the enrollment in the osteopathic colleges, and preosteopathic training of students. The Bureau of Professional Education and Colleges which met December 11 and 12 and the American Association of Osteopathic Colleges which met December 13 and 14 were also deeply concerned with the effects of the present national situation on osteopathic education. In other directions the reports from each of the colleges to the Board were very encouraging. The student body is at its maximum for the present capacity of the colleges which have been remarkably successful in meeting the demands resulting therefrom. The most serious problem is that of financing; the cost of educating osteopathic physicians and surgeons continues to increase.

The Bureau of Public Education on Health held meetings on December 12. It made several recommendations to the Association respecting laws pertaining to the public health.

The Board made the necessary small modifications of the Association's budget. The largest membership in the history of the Association was reported as well as the largest number in the profession. The report of the Bureau of Professional Education and Colleges added eight new residencies for a total of 148, one hospital to the list of Hospitals Approved for Resident Training for a total of thirty-four, five hospitals to the list of Hospitals Approved for Intern Training for a total of sixty-eight, and ten hospitals to the list of Registered Hospitals for a total of 134.

Dr. Robert E. Cole of Geneva, New York, resigned as program chairman for the 1952 convention because of additional duties undertaken on behalf of his divisional society of which he is secretary. Dr. William B. Strong, Brooklyn, was appointed by President-Elect Peckham to fill the vacancy.

The Board directed that a contract be drawn up for the completion of the interior of the third floor of the Andrew Taylor Still Memorial Building, needed for expansion of the services of the Association. The completed third floor will include a small auditorium, storage space, and additional offices. The necessary work includes flooring, ceiling, plastering, lighting, installation of partitions, and extension of heating and ventilation facilities.

The annual registration fee for certified osteopathic specialists was increased from five dollars to ten dollars, payable to the American Osteopathic Association. Five dollars of each fee will go to the various certifying boards to help cover secretarial costs and other expenses. In cases where an osteopathic physician is certified by two boards, each will receive two and a half dollars.

As the conference concluded and the President of the United States gave his emergency message to the nation, the Board directed that the following telegram be sent:

December 16, 1950

Honorable Harry S. Truman
White House
Washington, D. C.
Dear Mr. President:

The Board of Trustees of the American Osteopathic Association, in midyear session in Chicago, Illinois, on December 16, 1950, directed me as President to communicate to you complete assurance that the resources of the profession and its institutions, including physicians, surgeons, colleges and hospitals are available to you for service in this present emergency.

VINCENT P. CARROLL, D.O.
President, American Osteopathic Association

THE AUDRAIN COUNTY HOSPITAL CASE

A decision in the case involving a petition for a declaratory judgment filed by the board of trustees of the Audrain County Hospital, Mexico, Missouri, was rendered by Circuit Judge Sam C. Blair on November 29, 1950. The findings of fact and conclusions of law are set out in the columns of the Bureau of Public Education on Health which appear on pages 292 and 293 of this issue of THE JOURNAL.

These findings of fact and conclusions of law are important and of serious interest. The Court has held, in effect, in answer to the petition of the board of trustees of the Audrain County Hospital that the board has no right to exclude osteopathic physicians and surgeons from rendering services in the Hospital simply on the grounds that such doctors are adherents of a particular school of medicine.

The decision clearly indicates that, under the laws of Missouri, osteopathy is a school of medicine and

that at all times since 1897 the practice of osteopathy has included operative surgery with instruments and the use of drugs and that such have always been taught at the American School of Osteopathy and its successors at Kirksville, Missouri.

The board of trustees of the Audrain County Hospital asked for clarification of the law pertaining to county hospitals so that it might carry out its duties in conformity with the law. The findings of fact and conclusions of law relating to the character of osteopathic practice were rendered as a result of issues raised by doctors of medicine in Audrain County and the Missouri State Medical Association who had been named as defendants in the petition.

The findings of the Court leave little to the imagination. Whether or not the case will be appealed to a higher court is not known at this time. The decision of the Circuit Court is obviously justified by the clear and unmistakable evidence presented at the trial.

R. C. McCaughan, D.O.
Executive Secretary

MIDCENTURY WHITE HOUSE CONFERENCE ON CHILDREN AND YOUTH

The Midcentury White House Conference on Children and Youth, held in Washington December 3-7 and attended by some 6,000 specialists in the field of childhood and youth, brought together widely varying groups who have responsibilities for the care and training of the preadult segments of the population. Probably no other civilization has ever approached the successful organized efforts of these people to improve the conditions of childhood and youth and certainly none has been so fortunate in ability to provide the necessary facilities for the complete development of the capabilities of youth—and still there is a long way to go. Recognizing this, the President of the United States invited leaders among those who devote their lives to the problems of young people to Washington last month for the fifth White House Conference. The first such Conference was held in 1909. Others were held in 1919, 1930, and 1940.

Out of the studies of this last Conference, at which the osteopathic profession was well represented, grew a considerable number of conclusions and objectives which ended with a "Pledge to Children." The Pledge, with all its significance, follows. It is commended to the attention of every practicing physician.

PLEDGE TO CHILDREN

As its closing action, the Conference unanimously adopted the following pledge.

TO YOU, our children, who hold within you our most cherished hopes, we the members of the Midcentury White House Conference on Children and Youth, relying on your full response, make this pledge:

From your earliest infancy we give you our love, so that you may grow with trust in yourself and in others.

We will recognize your worth as a person and we will help you to strengthen your sense of belonging.

We will respect your right to be yourself and at the same time help you to understand the rights of others, so that you may experience cooperative living.

We will help you to develop initiative and imagination, so that you may have the opportunity freely to create.

We will encourage your curiosity and your pride in workmanship, so that you may have the satisfaction that comes from achievement.

We will provide the conditions for wholesome play that will add to your learning, to your social experience, and to your happiness.

We will illustrate by precept and example the value of integrity and the importance of moral courage.

We will encourage you always to seek the truth.

We will provide you with all opportunities possible to develop your own faith in God.

We will open the way for you to enjoy the arts and to use them for deepening your understanding of life.

We will work to rid ourselves of prejudice and discrimination, so that together we may achieve a truly democratic society.

We will work to lift the standard of living and to improve our economic practices, so that you may have the material basis for a full life.

We will provide you with rewarding educational opportunities, so that you may develop your talents and contribute to a better world.

We will protect you against exploitation and undue hazards and help you grow in health and strength.

We will work to conserve and improve family life and, as needed, to provide foster care according to your inherent rights.

We will intensify our search for new knowledge in order to guide you more effectively as you develop your potentialities.

As you grow from child to youth to adult, establishing a family life of your own and accepting larger social responsibilities, we will work with you to improve conditions for all children and youth.

Aware that these promises to you cannot be fully met in a world at war, we ask you to join us in a firm dedication to the building of a world society based on freedom, justice and mutual respect.

SO MAY YOU grow in joy, in faith in God and in man, and in those qualities of vision and of the spirit that will sustain us all and give us new hope for the future.

R. C. McCaughan, D.O.

THE EDUCATIONAL SUPPLEMENT

This Journal contains the Educational Supplement, contributed by the Office of Education of the American Osteopathic Association. Through the years much material on osteopathic education has been published in The Journal, but an Educational Supplement was not undertaken until January of 1949. The outstanding usefulness of that Supplement and of the one published in January of 1950 have provided convincing proof that the undertaking should be repeated yearly. Mr. Lawrence W. Mills, Director of the Office of Education, has compiled material which should not only be helpful and informative to those interested in osteopathic education from any angle, but also it should be of interest to every member of the profession.

The statistical tables show progress in many directions which should be a source of pride. The material concerning objectives of osteopathic education, selection of students, scholarships, accreditation of colleges, and intern training should be studied by every osteopathic physician. The complete text of "Educational Standards for Osteopathic Colleges," which is included, adds to the value of the Supplement.